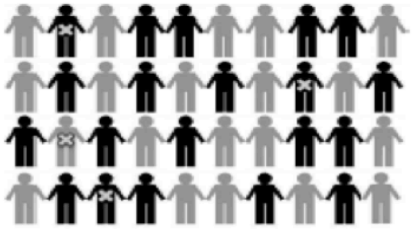
New directions in population health science: from risks to consequences

Sandro Galea

Boston University School of Public Health

1. What we do



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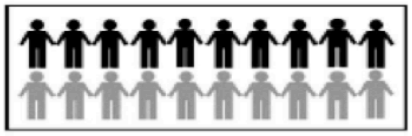
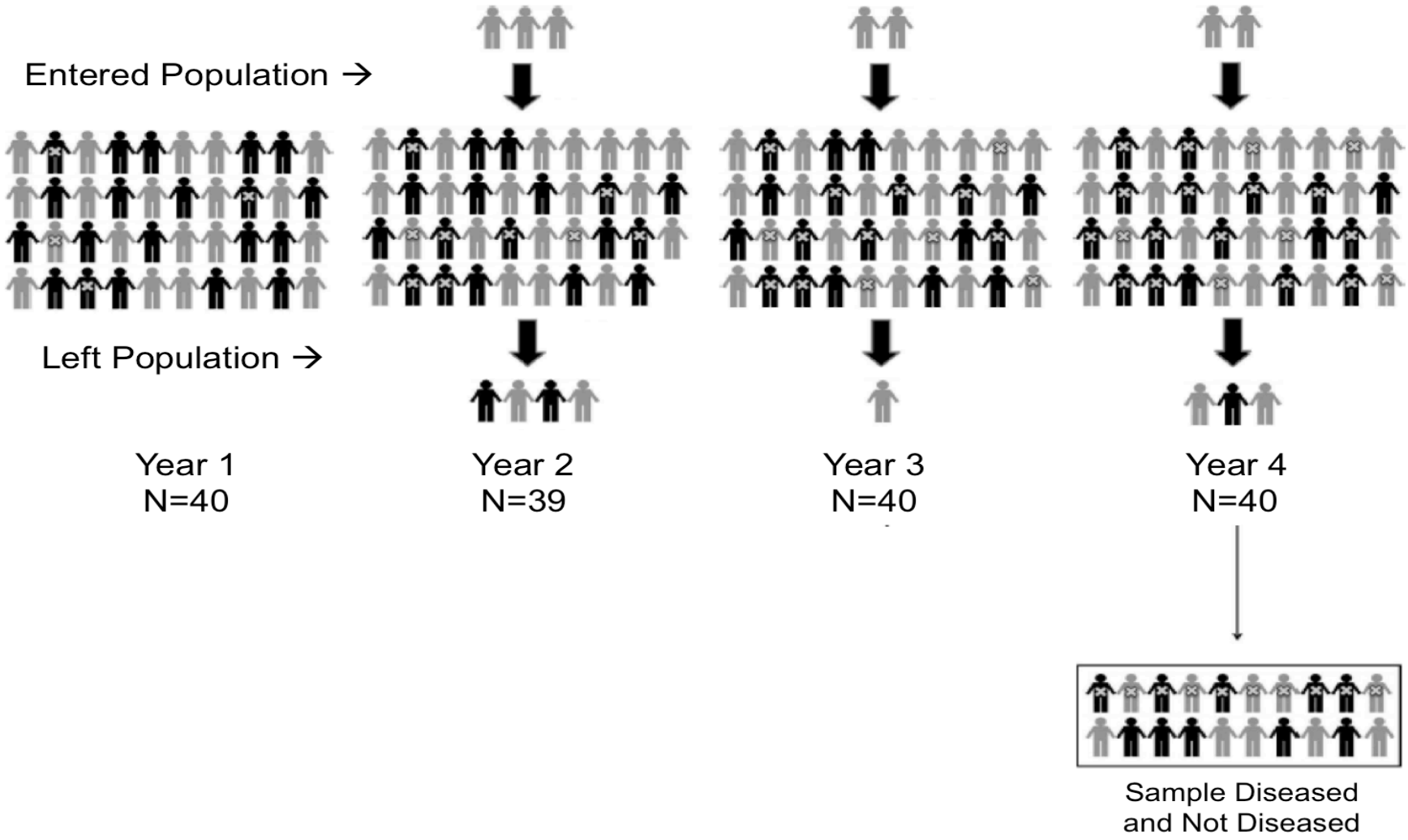
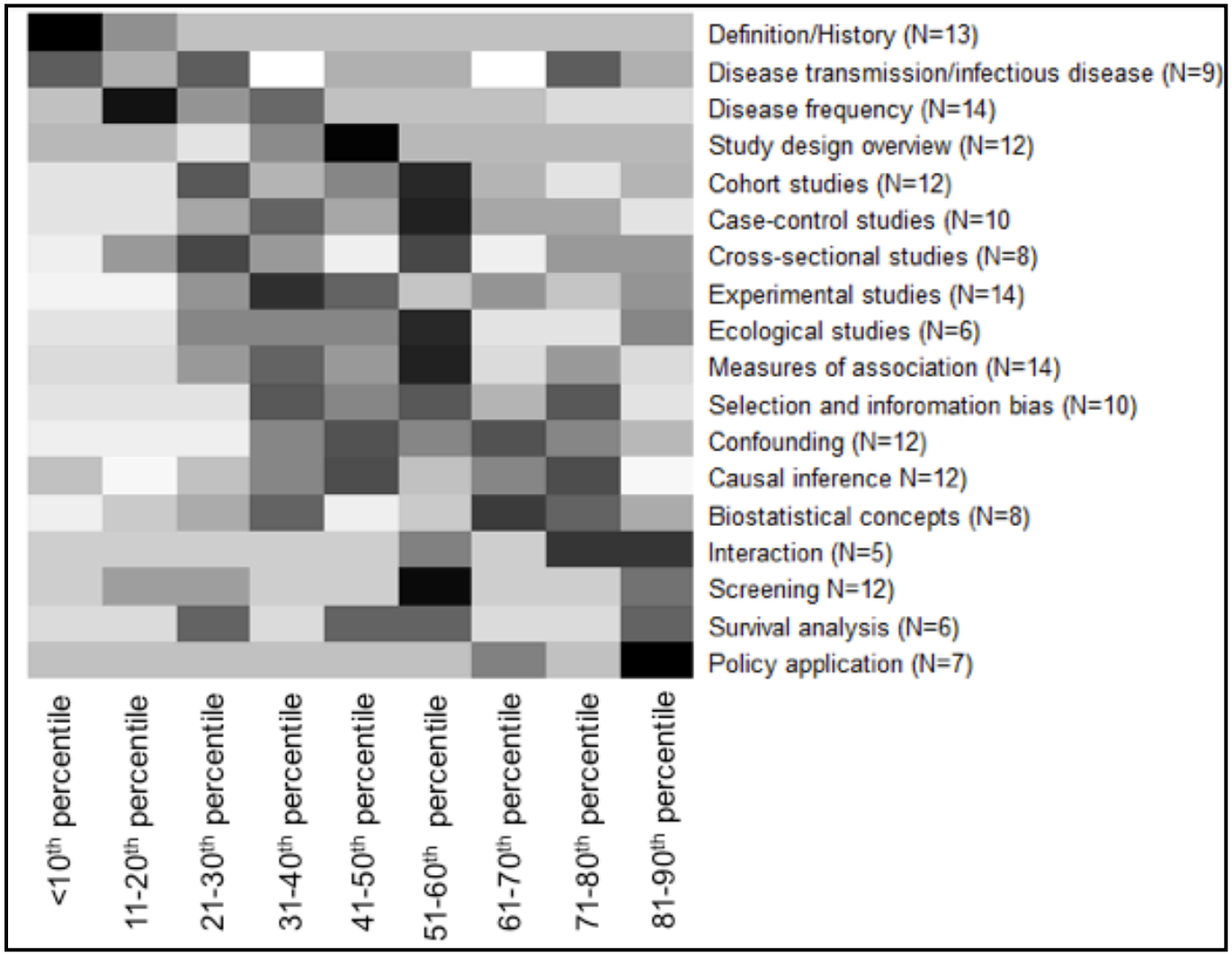


Sample Exposed
and Unexposed

	Diseased	Not Diseased	**Total**
Exposed	8	2	10
Unexposed	5	5	10
Total	13	7	20



Keyes KM, Galea S. Epidemiology Matters. Oxford University Press. 2014. Chapter 4

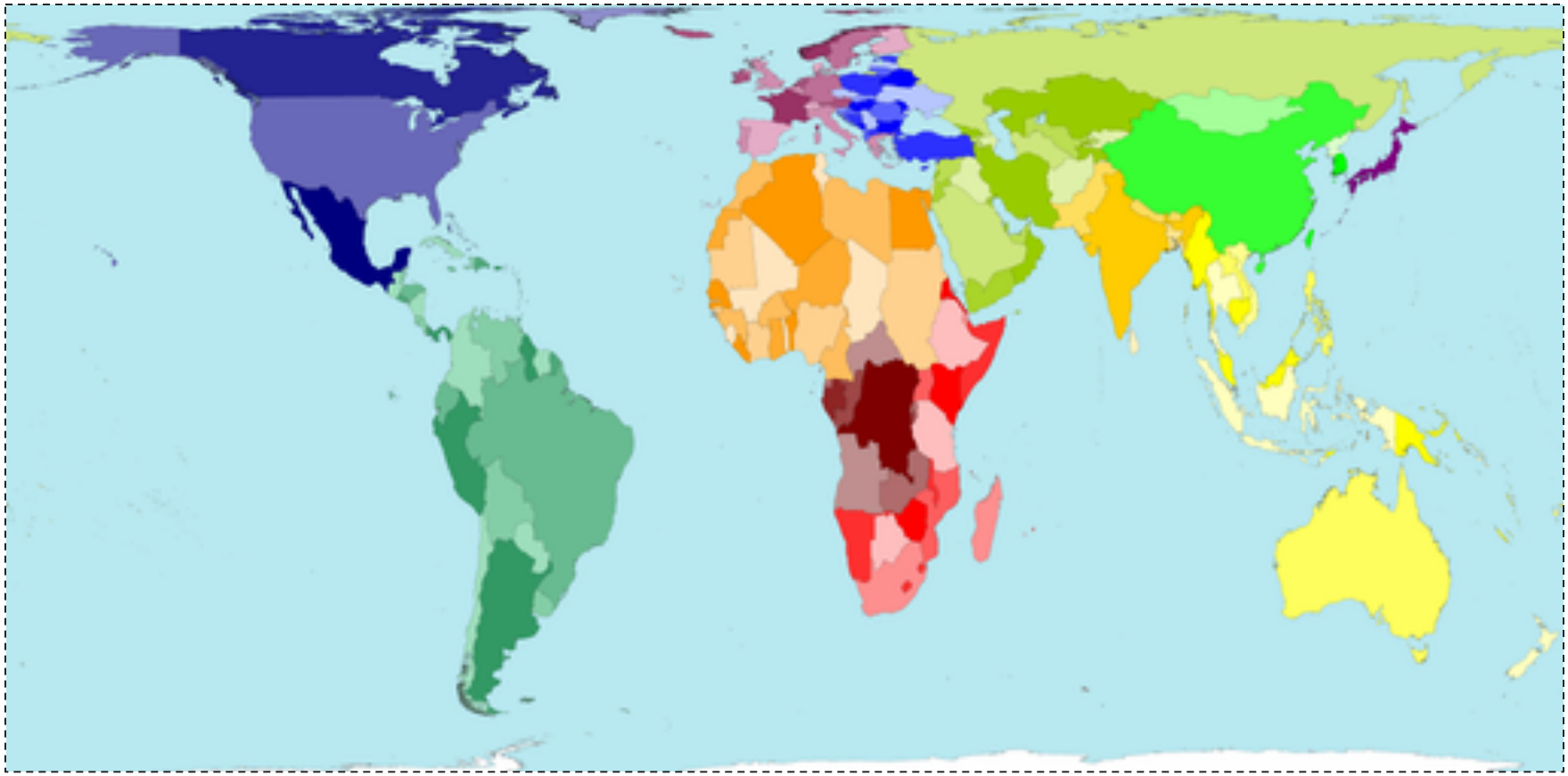


Keyes KM, Galea S. (2014). Current practices in teaching introductory epidemiology: how we got here, where to go. American Journal of Epidemiology, 180(7): 661-668. PMID: 25190677.

2. What this leads to

a. The state of the world’s health

The world, actual size



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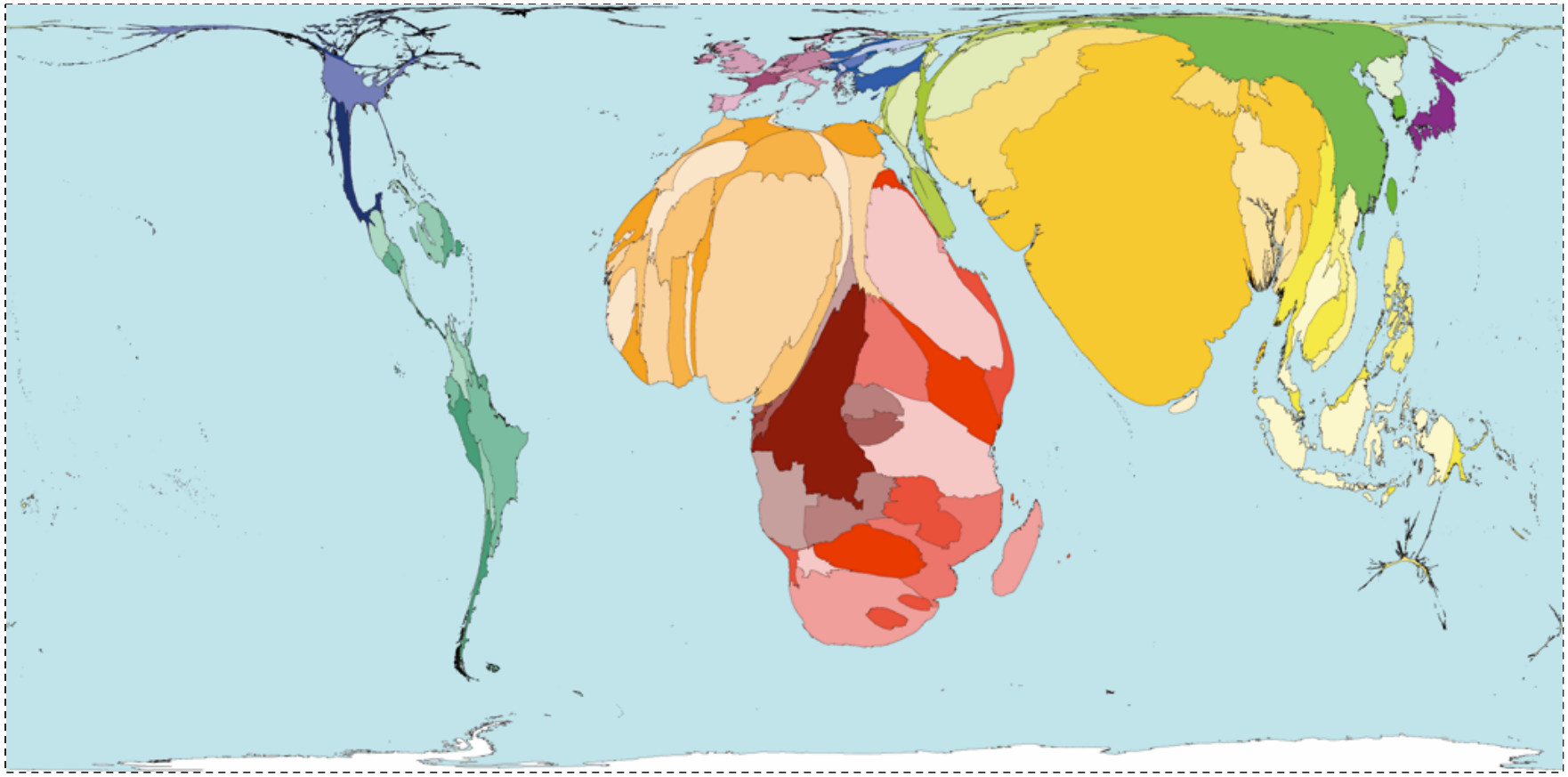
The world, by preventable deaths



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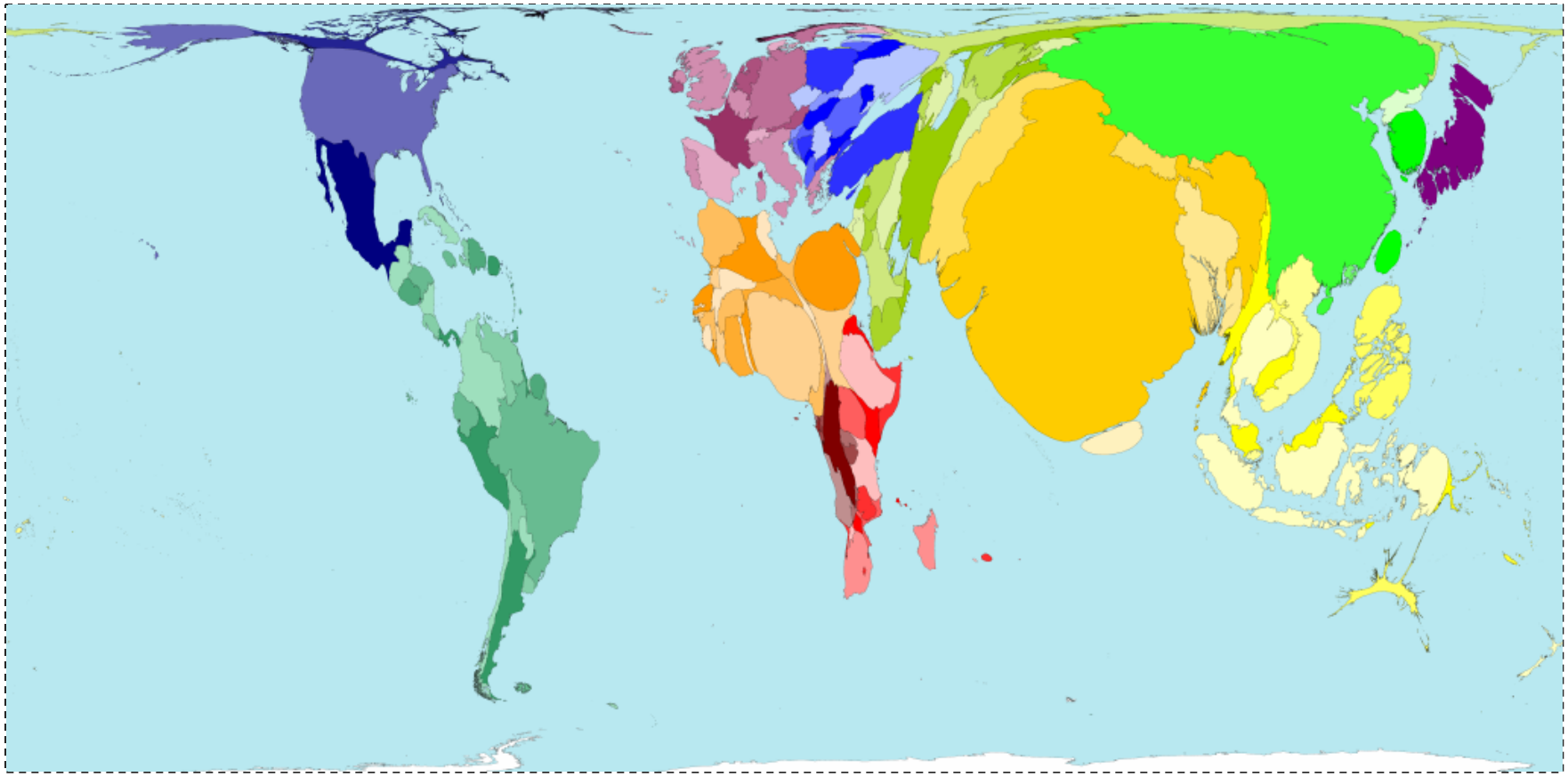
The world, by unhealthy life expectancy



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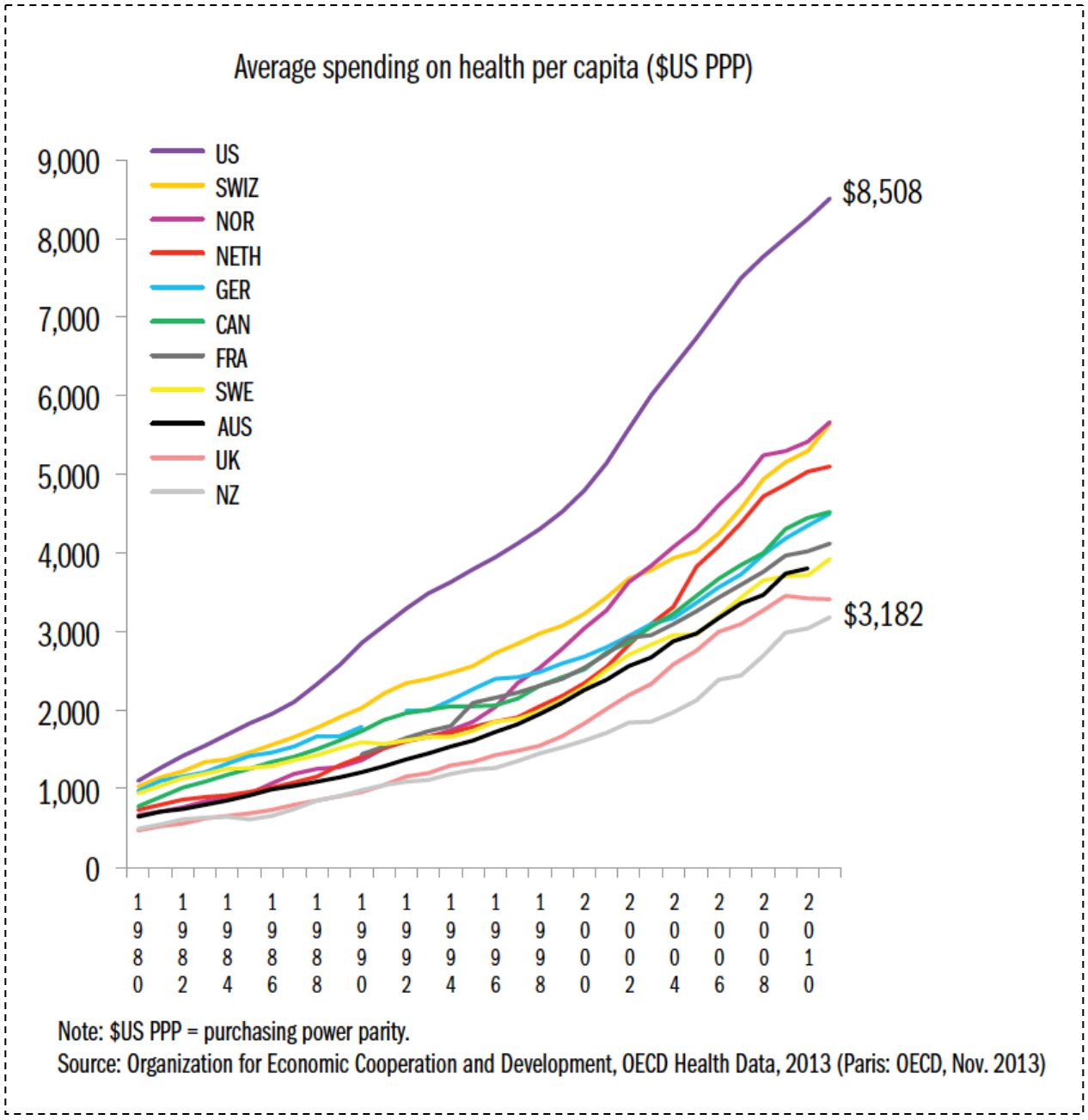


http://www.commonwealthfund.org/~/media/files/publications/fund-report/2014/jun/1755_davis_mirror_mirror_2014.pdf

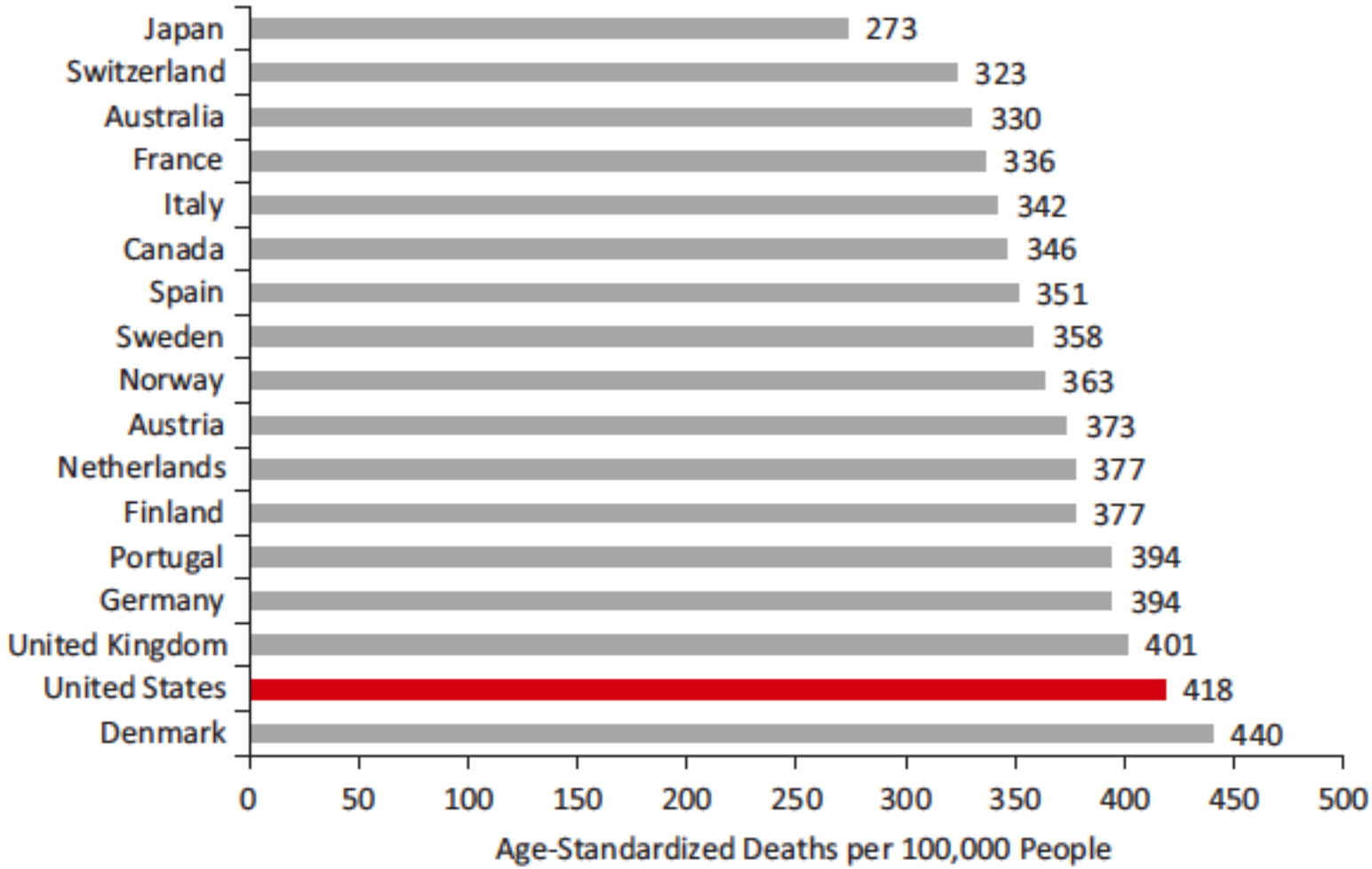


FIGURE 1-1 Mortality from noncommunicable diseases in 17 peer countries, 2008.
SOURCE: Data from World Health Organization (2011a, Table 3).

US Health in International Perspective. Shorter lives, poorer health. S Woolf, L Aron, eds. NRC and IOM. 2012.

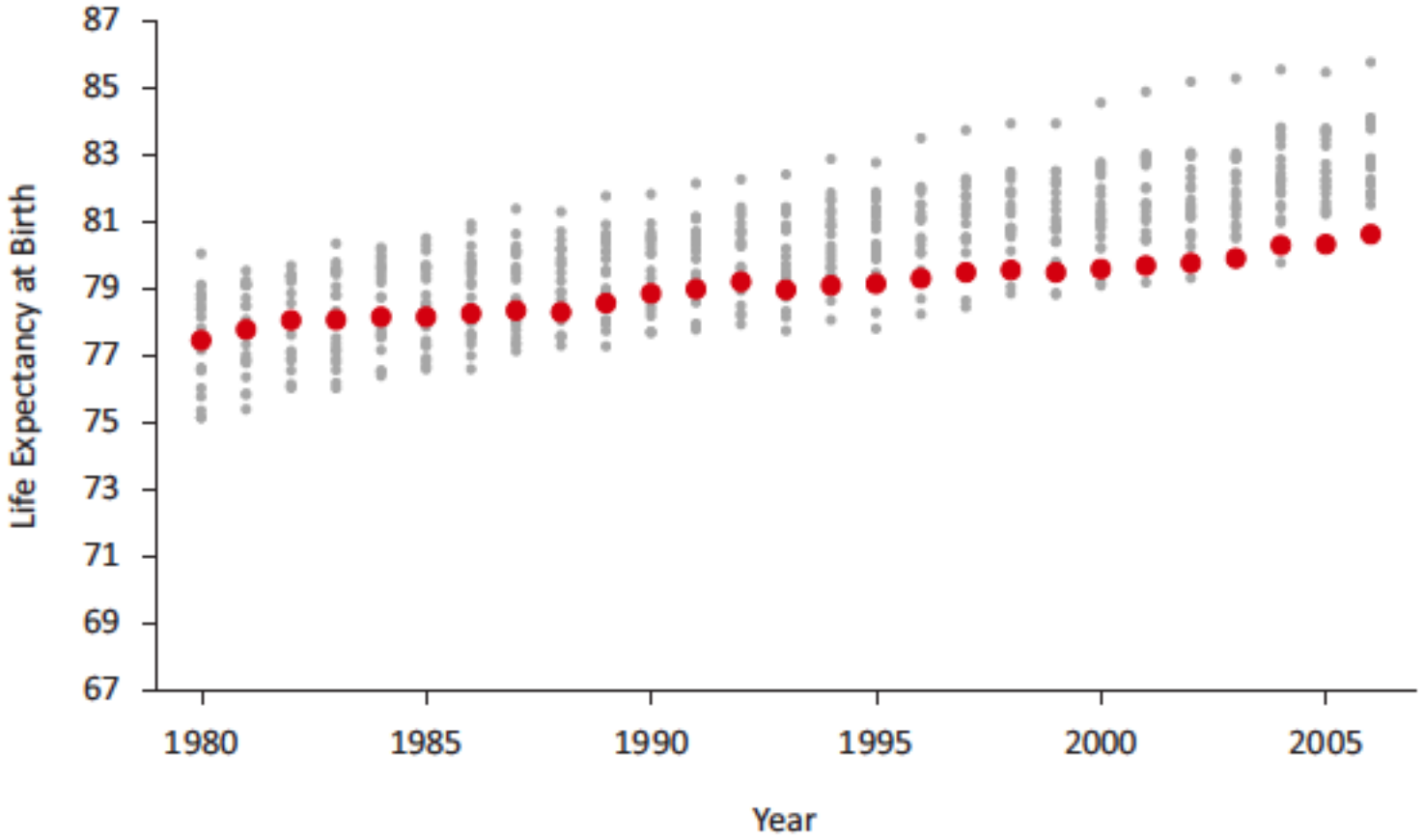


FIGURE 1-6 U.S. female life expectancy at birth relative to 21 other high-income countries, 1980-2006.

NOTES: Red circles depict newborn life expectancy in the United States. Grey circles depict life expectancy values for Australia, Austria, Belgium, Canada, Denmark, Finland, France, Iceland, Ireland, Italy, Japan, Luxembourg, the Netherlands, New Zealand, Norway, Portugal, Spain, Sweden, Switzerland, the United Kingdom, and West Germany.

SOURCE: National Research Council (2011, Figure 1-4).

US Health in International Perspective. Shorter lives, poorer health. S Woolf, L Aron, eds. NRC and IOM. 2012.

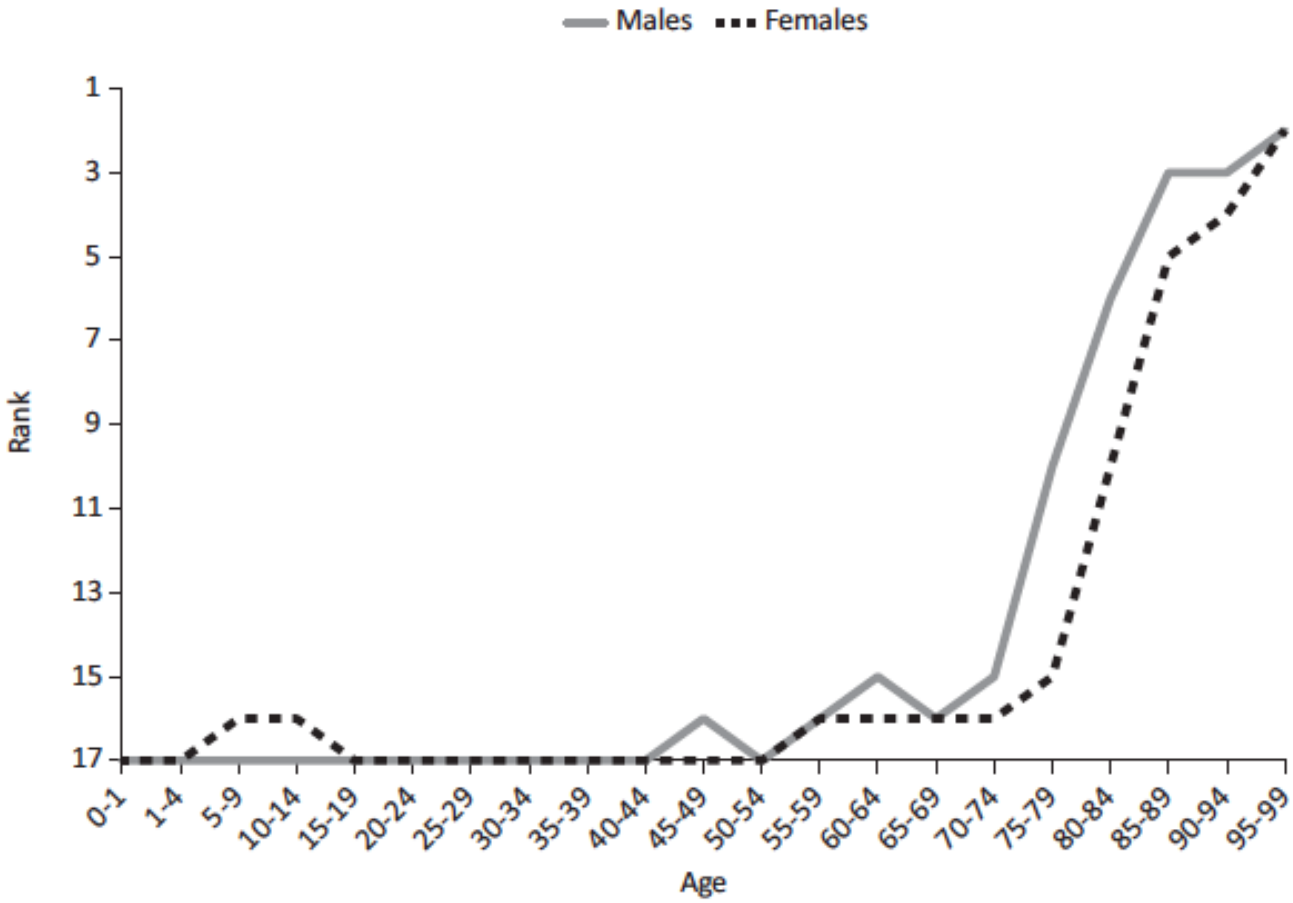
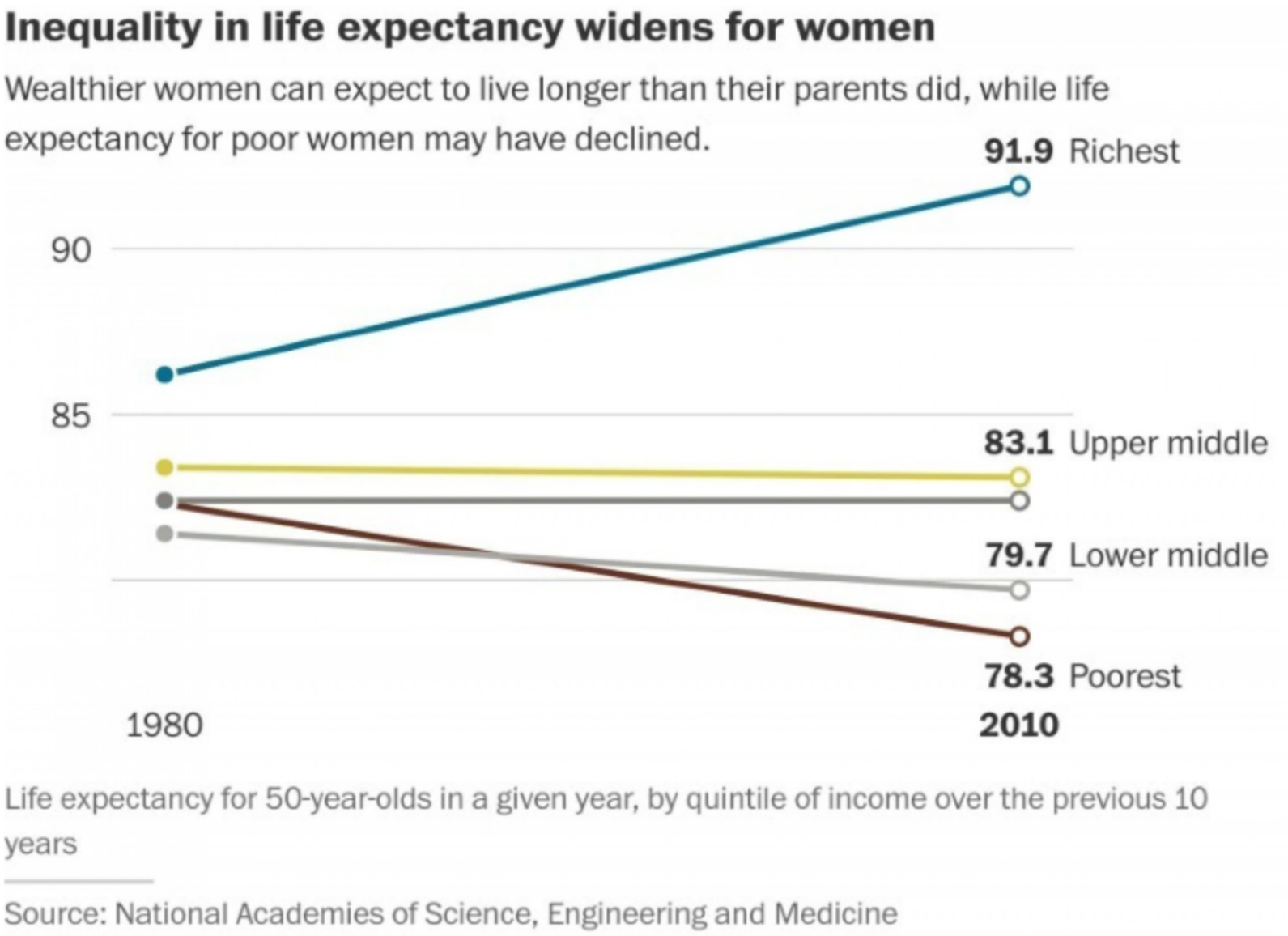


FIGURE 1-9 Ranking of U.S. mortality rates, by age group, among 17 peer countries, 2006-2008.

NOTES: The top rank is number 1, indicating the lowest death rate, and the bottom rank is number 17, indicating the highest death rate. Rankings are based on all-cause mortality rates for 2006-2008. Data for this figure were drawn from (1) the Human Mortality Database, 2011, University of California, Berkeley (USA), and Max Planck Institute for Demographic Research (Germany), available at http://www.mortality.org or http://www.humanmortality.de (data downloaded July 18, 2011) and (2) Arias, Elizabeth, 2011, United States Life Tables, 2007. *National Vital Statistics Reports, 59*(9), Hyattsville, MD: National Center for Health Statistics.

SOURCE: Adapted from Ho and Preston (2011, Figure 1).

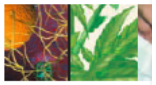


National Academy of Medicine. The growing gap in life expectancy by income: Implications for federal programs and policy responses. 2015. http://www.nap.edu/download.php?record_id=19015#

b. A paradoxical approach to this state of the world

Revista Española de Enfermedades Digestivas

Rev Esp Enferm D
Vol. 102, N.° 10, pp. 573-



The NE

Personalized medicine: inevitable

Perspective

JULY 22, 2010

The Path to Personalized Medicine

Margaret A. Hamburg, M.D., and Francis S. Collins, M.D., Ph.D.

Major investments in basic science have created
an opportunity for significant progress in
clinic
hund

the NIH and the FDA will invest
in advancing translational and

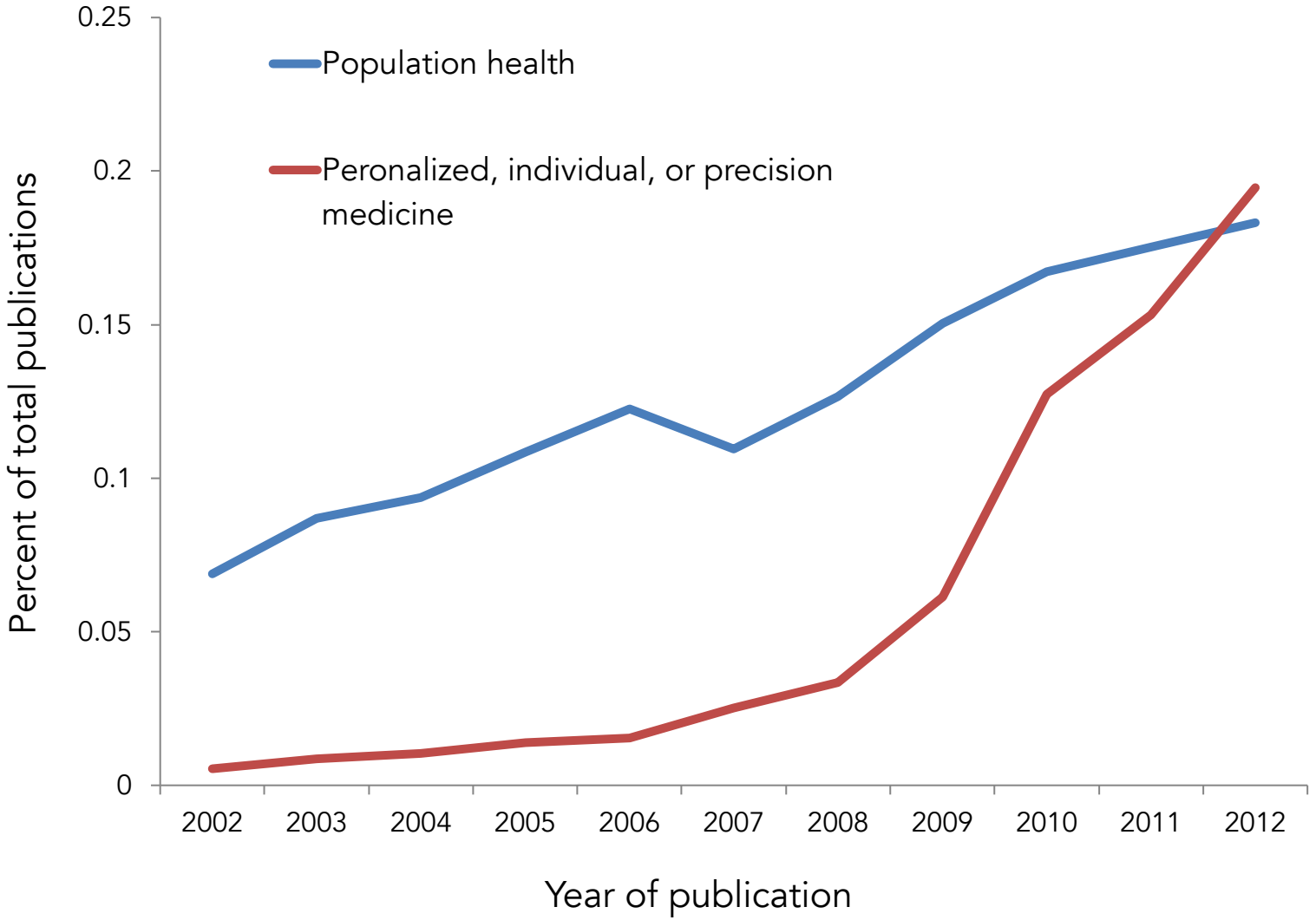
Journal of Diabetes Science and Technology
Volume 3, Issue 4, July 2009

SYMPOSIUM

The Case for Personalized Medicine

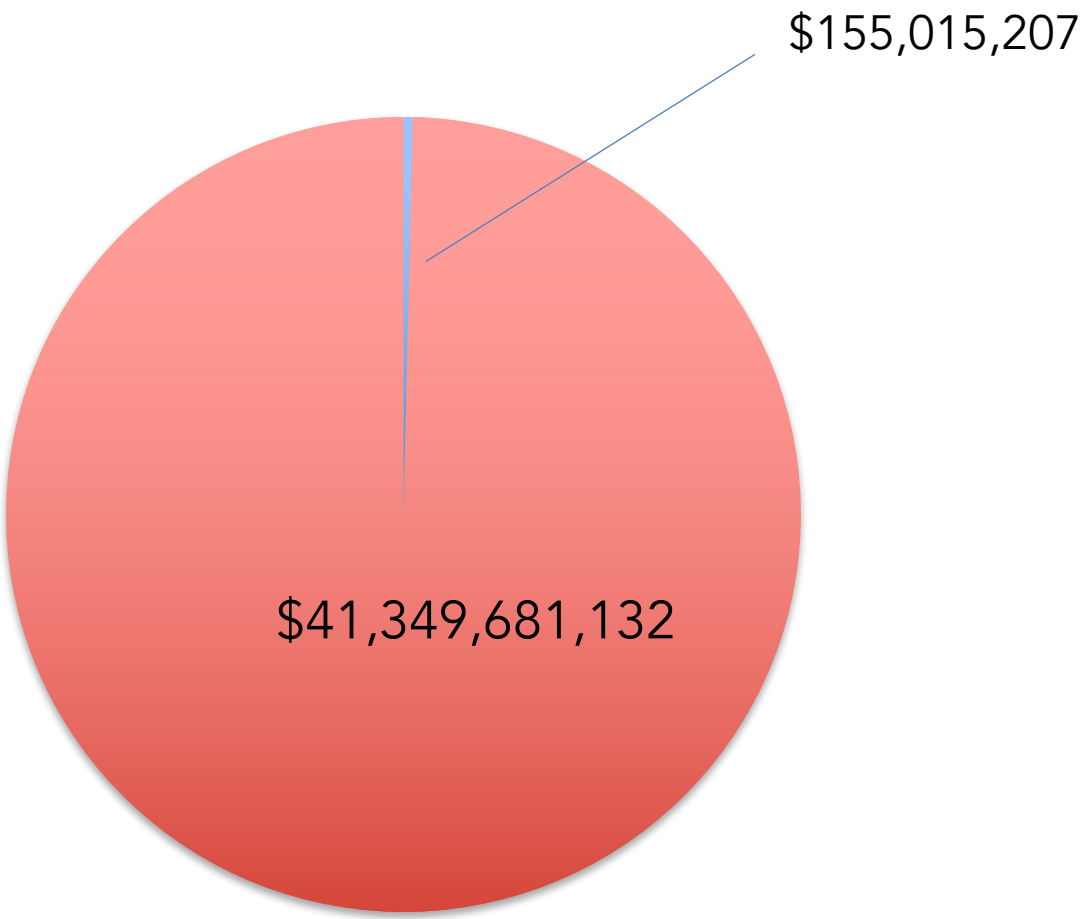
Edward Abrahams, Ph.D.,[1] and Mike Silver, Ph.D.[2]

Proportion of papers in PubMed, 2002 - 2012



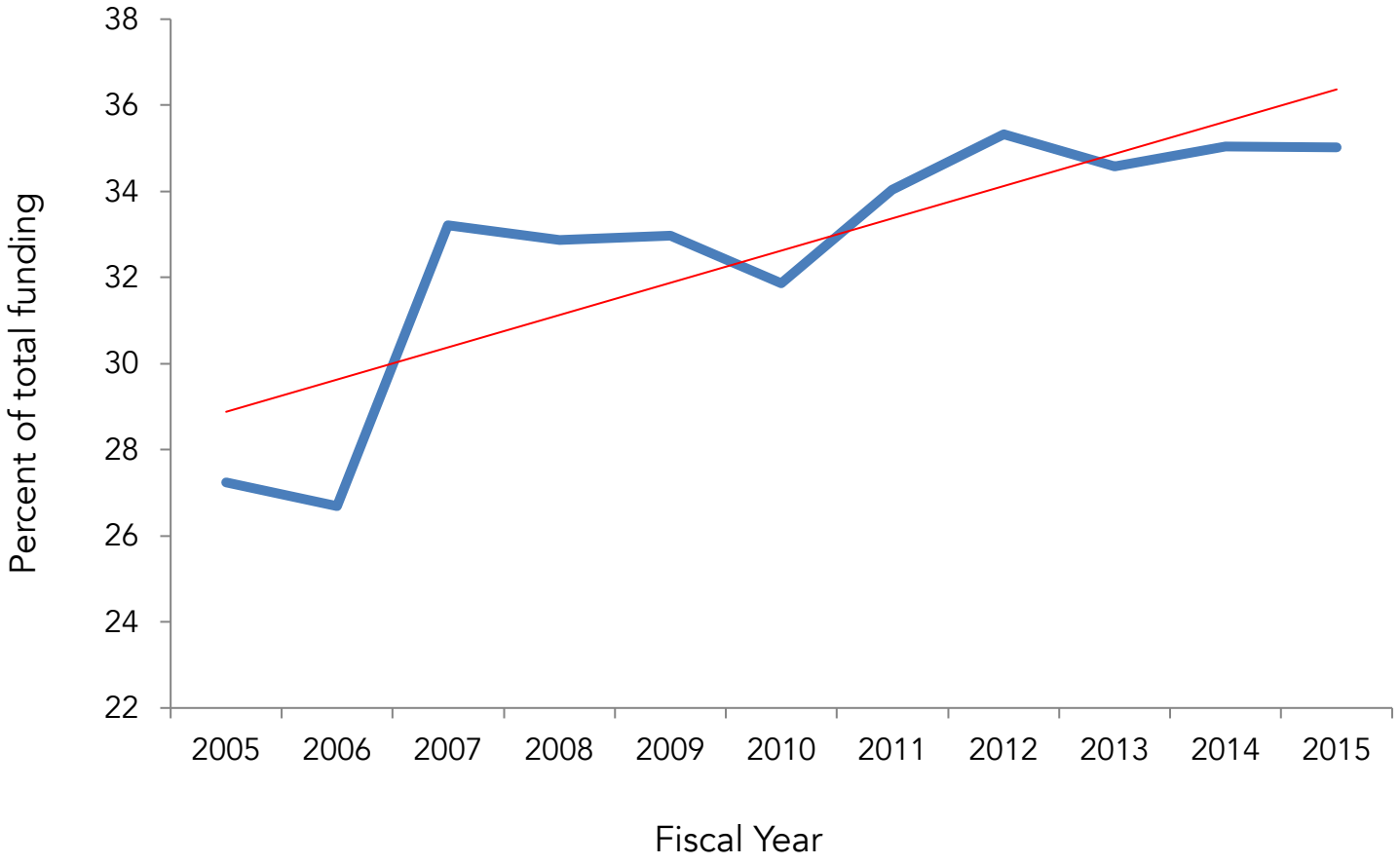
PubMed search results for “population health” and "Individualized Medicine" or "personalized medicine" or "personalised medicine" or “individual medicine” or “precision medicine” from 2002-2012. <http://www.ncbi.nlm.nih.gov/> Accessed November 20, 2014.

Among NIH funding for the current fiscal year, only 0.4% was awarded to projects with the terms “population” or “public” in the title



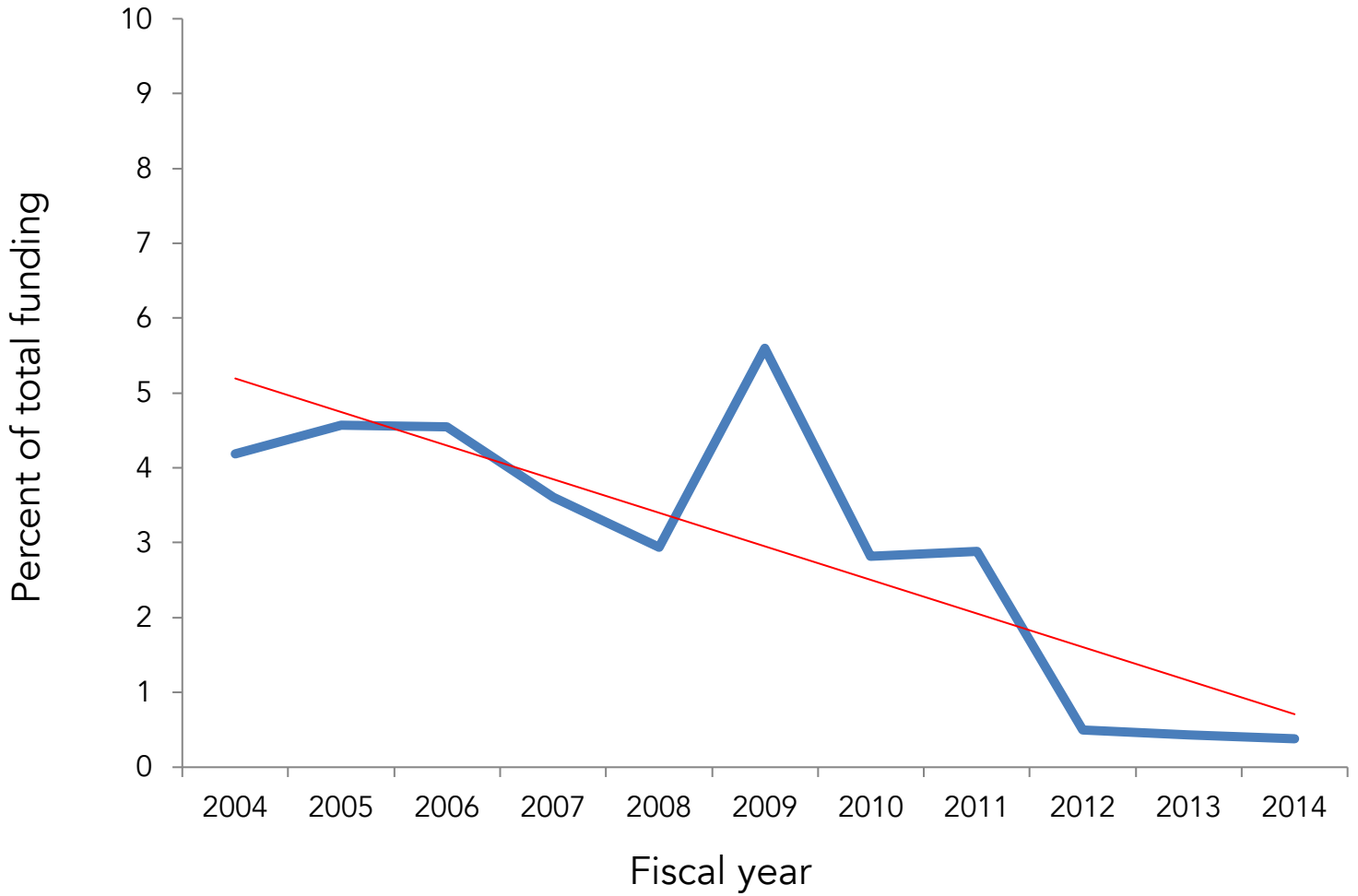
NIH RePORTER. Current projects from FY 2013-2014 for which funding data is available. <http://projectreporter.nih.gov/reporter_ChartResults.cfm?icde=22550343> Accessed on November 19, 2014.

Proportion of NIH funding awarded to projects with the terms “genetic” or “genetics” in the title, abstract or terms



NIH RePORTER. Search results for projects for which funding data is available. <http://projectreporter.nih.gov/reporter.cfm> Accessed on November 20, 2014.

Proportion of NIH funding awarded to projects with the terms "population" or "public" in the title, abstract, or terms



NIH RePORTER. Search results for projects for which funding data is available. <http://projectreporter.nih.gov/reporter.cfm> Accessed on November 20, 2014.

The impact of communicating genetic risks of disease on risk-reducing health behaviour: systematic review with meta-analysis

Gareth J Hollands,[1] David P French,[2] Simon J Griffin,[3] A Toby Prevost,[4] Stephen Sutton,[3] Sarah King,[1] Theresa M Marteau[1]

[1]Behaviour and Health Research Unit, University of Cambridge, Cambridge, UK
[2]School of Psychological Sciences, University of Manchester, Manchester, UK
[3]Department of Public Health and Primary Care, University of Cambridge, Cambridge, UK
[4]Imperial Clinical Trials Unit, Imperial College London, London, UK

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Additional material is published online only. To view please visit the journal online.

ABSTRACT

OBJECTIVE
To assess the impact of communicating DNA based disease risk estimates on risk-reducing health behaviours and motivation to engage in such behaviours.

DESIGN
Systematic review with meta-analysis, using Cochrane methods.

DATA SOURCES
Medline, Embase, PsycINFO, CINAHL, and the Cochrane Central Register of Controlled Trials up to 25 February 2015. Backward and forward citation searches were also conducted.

(standardised mean difference 0.12, 95% confidence interval −0.00 to 0.24, P=0.05), or physical activity (standardised mean difference −0.03, 95% confidence interval −0.13 to 0.08, P=0.62). There were also no effects on any other behaviours (alcohol use, medication use, sun protection behaviours, and attendance at screening or behavioural support programmes) or on motivation to change behaviour, and no adverse effects, such as depression and anxiety. Subgroup analyses provided no clear evidence that communication of a risk-conferring genotype affected behaviour more than communication of the absence of such a genotype. However, studies were predominantly at high or unclear risk of bias, and evidence was typically of low quality.

CONCLUSIONS
Expectations that communicating DNA based risk estimates changes behaviour is not supported by existing evidence. These results do not support use of genetic testing or the search for risk-conferring gene

Expectations that communicating DNA based risk estimates changes behaviour is not supported by existing evidence

WHAT IS ALREADY KNOWN ON THIS TOPIC

Genetic testing is being increasingly used in a growing number of healthcare settings and in direct-to-consumer testing for a range of common complex disorders

There is an expectation that communicating DNA based disease risk estimates will motivate changes in key health behaviours, including smoking, diet, and physical activity

There is a need for a rigorous systematic review to examine whether communicating genetic risks does indeed motivate risk-reducing behaviour change

WHAT THIS STUDY ADDS

The results of this updated systematic review with meta-analysis using Cochrane methods suggest that communicating DNA based disease risk estimates has little or no impact on risk-reducing health behaviour

Existing evidence does not support expectations that such interventions could play a major role in motivating behaviour change to improve population health

realised through communicating the results of such predictions. For example, does communicating to smokers that they have an increased genetic risk of developing lung cancer motivate smoking cessation, or does telling middle aged people that they have an increased genetic risk of developing diabetes motivate increased physical activity to reduce this risk? These are particularly timely questions, given high levels of interest in personalised medicine and in direct-to-consumer testing. More than 10 years ago, direct-to-consumer tests for a range of common complex disorders were rushed to market. These tests continue to be sold in Canada, the United Kingdom, and other European countries, including Denmark, Finland, the Netherlands, Sweden, and Ireland (www.23andme.com/en-gb/health/; www.23andme.com/en-eu/), with continued international expansion likely. In the United States, expansion was tempered in 2013 when the Food and Drug Administration ordered the company 23andme to stop selling its testing kits because of concerns about their accuracy and usefulness, but as of October 2015 the company has resumed selling some

ARTICLE

doi:10.1038/nature18642

The genetic architecture of type 2 diabetes

A list of authors and affiliations appears in the online version of the paper

The genetic architecture of common traits, including the number, frequency, and effect sizes of inherited variants that contribute to individual risk, has been long debated. Genome-wide association studies have identified scores of common variants associated with type 2 diabetes, but in aggregate, these explain only a fraction of the heritability of this disease. Here, to test the hypothesis that lower-frequency variants explain much of the remainder, the GoT2D and T2D-GENES consortia performed whole-genome sequencing in 2,657 European individuals with and without diabetes, and exome sequencing in 12,940 individuals from five ancestry groups. To increase statistical power, we expanded the sample size via genotyping and imputation in a further 111,548 subjects. Variants associated with type 2 diabetes after sequencing were overwhelmingly common and most fell within regions previously identified by genome-wide association studies. Comprehensive enumeration of sequence variation is necessary to identify functional alleles that provide important clues to disease pathophysiology, but large-scale sequencing does not support the idea that lower-frequency variants have a major role in predisposition to type 2 diabetes.

There is compelling evidence that the individual risk of type 2 diabetes

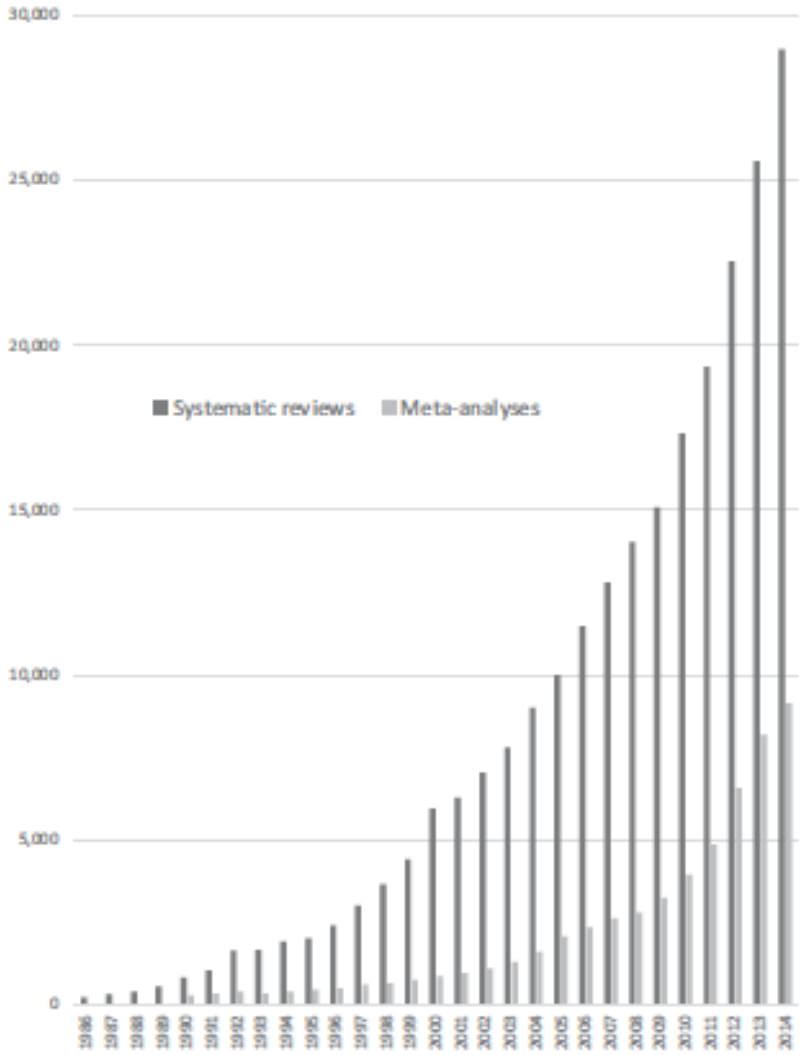
Comprehensive enumeration of sequence variation is necessary to identify functional alleles that provide important clues to disease pathophysiology, but large-scale sequencing does not support the idea that lower-frequency variants have a major role in predisposition to type 2 diabetes.

cases of type 2 diabetes (T2D) and ancestry-matched normoglycaemic controls from northern and central Europe (Methods and Supplementary Table 1). To increase power to identify low-frequency ($0.5\% < MAF < 5\%$) and rare ($MAF < 0.5\%$) T2D variants with large effects, we preferentially identified individuals from the extremes of genetic risk (Methods). The genome sequence of 1,326 cases and 1,331 control individuals was determined through joint statistical analysis of low-coverage whole-genome sequence (~5×), deep-coverage exome sequence (~82×), and array-based genotypes at 2.5 million (Methods).

Power to detect association with low-frequency and rare variants of modest effect is limited in a sample of 2,657 individuals. To increase power for variants discovered via genome sequencing, we imputed sequence-based genotypes into 44,414 additional individuals of European origin (11,645 T2D cases and 32,769 controls; Methods) from 13 studies (Supplementary Table 3). We estimated power in the combined sequence plus imputed data, adjusting for imputation

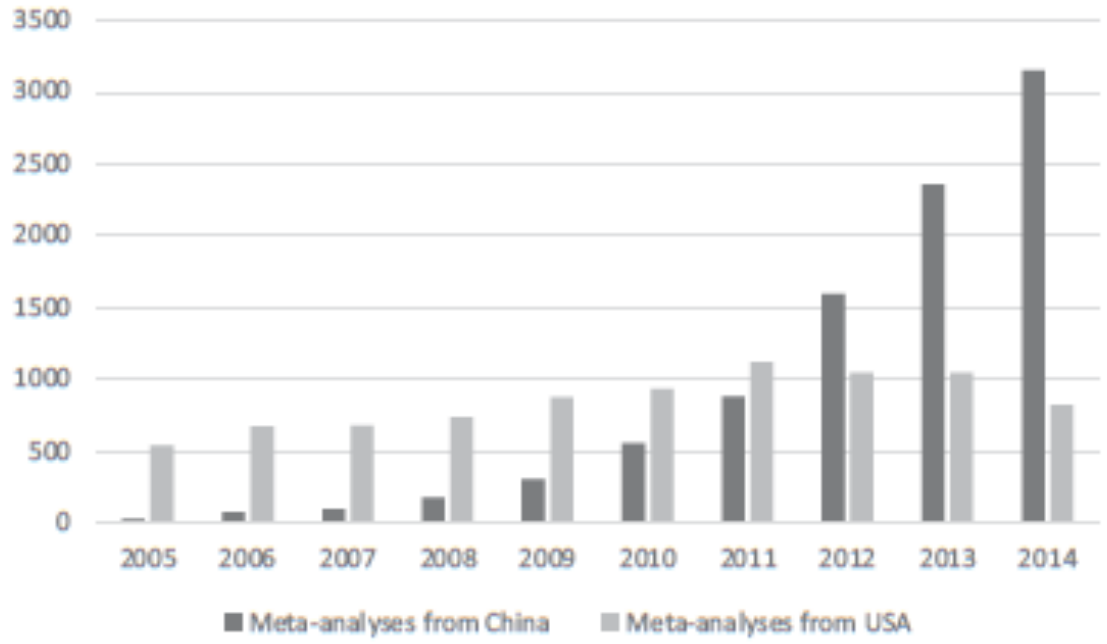
c. A somewhat shambling scientific approach

Figure 1. Number of PubMed-Indexed Articles Published Each Year Between 1986 and 2014 That Carry the Tag "Systematic Review" or "Meta-analysis" for Type of Publication



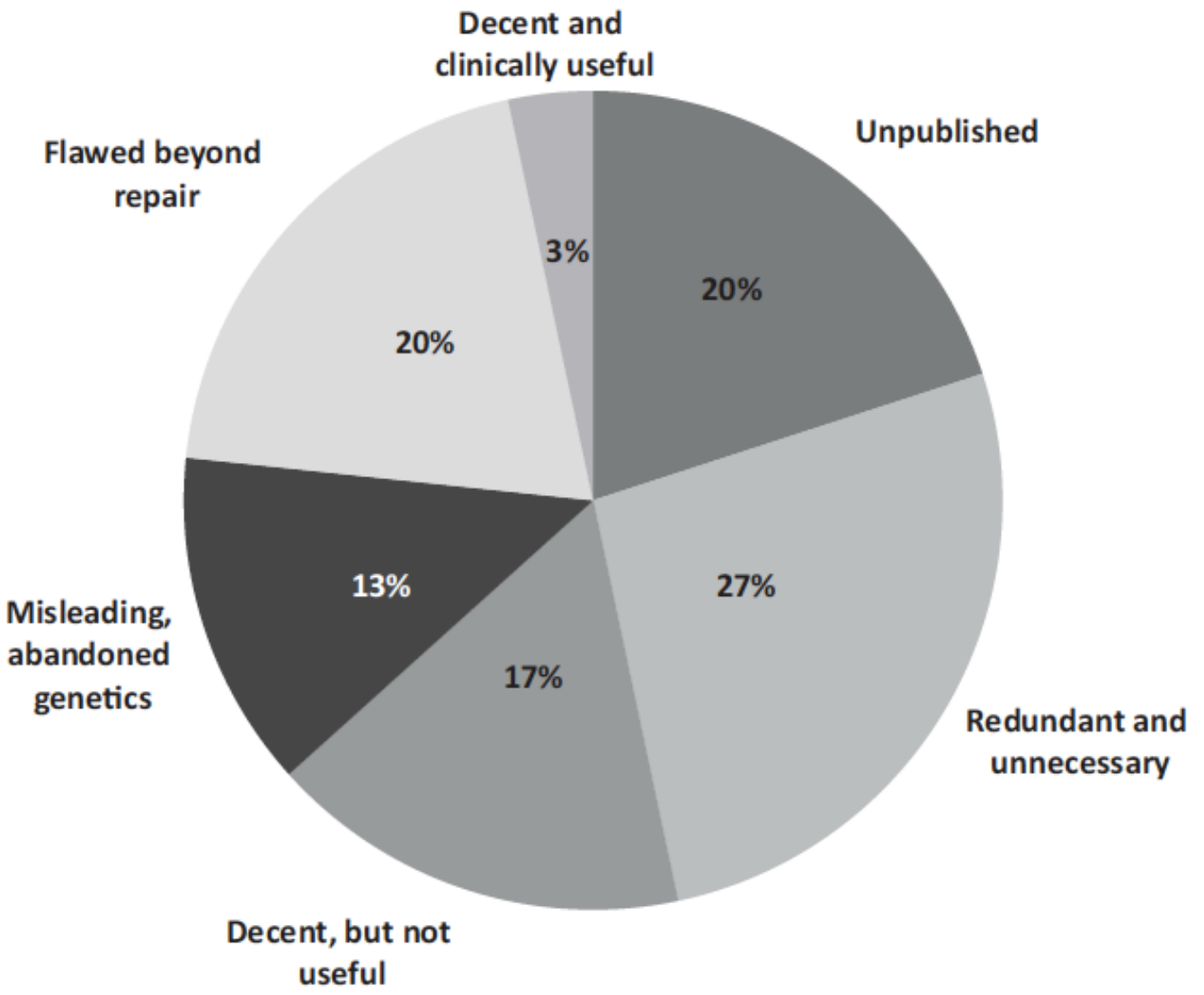
Ionnidis JPA. The Mass Production of Redundant, Misleading, and Conflicted Systematic Reviews and Meta-analyses. Milbank Quarterly. Vol. 94, No. 3, 2016 (pp. 485-514)

Figure 2. Number of PubMed-Indexed Articles Published Each Year Between 2005 and 2014 That Carry the Tag "Meta-analysis" for Type of Publication and Have Author Affiliations From China or From the United States (USA)



Ionnidis JPA. The Mass Production of Redundant, Misleading, and Conflicted Systematic Reviews and Meta-analyses. Milbank Quarterly. Vol. 94, No. 3, 2016 (pp. 485-514)

Figure 4. A Summary Overview of Currently Produced Meta-analyses



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What types of interventions generate inequalities? Evidence from systematic reviews

Theo Lorenc,[1] Mark Petticrew,[1] Vivian Welch,[2] Peter Tugwell[2]

► An additional appendix is published online only. To view this file please visit the journal online (http://dx.doi.org/10.1136/jech-2012-201257).

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[2]Centre for Global Health, University of Ottawa, Ottawa, Canada

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ABSTRACT

Background Some effective public health interventions may increase inequalities by disproportionately benefiting less disadvantaged groups ('intervention-generated inequalities' or IGIs). There is a need to understand which types of interventions are likely to produce IGIs, and which can reduce inequalities.

Methods We conducted a rapid overview of systematic reviews to identify evidence on IGIs by socioeconomic status. We included any review of non-healthcare interventions in high-income countries presenting data on differential intervention effects on any health status or health behaviour outcome. Results were synthesised narratively.

Results The following intervention types show some evidence of increasing inequalities (IGIs) between socioeconomic status groups: media campaigns; and workplace smoking bans. However, for many intervention types, data on potential IGIs are lacking. By contrast, the following show some evidence of reducing health inequalities: structural workplace interventions; provision of resources; and fiscal interventions, such as tobacco pricing.

Conclusion Our findings are consistent with the idea that 'downstream' preventive interventions are more likely to increase health inequalities than 'upstream' interventions. More consistent reporting of differential intervention effectiveness is required to help build the evidence base on IGIs.

INTRODUCTION

A number of researchers have raised concerns about the possibility that public health interventions may increase inequalities in the population. This has been expressed as an 'inverse prevention law',[1] analogous to the 'inverse care law' posited for medical care by Tudor Hart,[2] stating that those in most need of benefiting from preventive interventions are least likely to receive them. That is, even where interventions are successful at improving health across the population, they may increase health inequalities. This can happen where an intervention is of greater benefit to advantaged (lower-risk) groups than to disadvantaged (higher-risk) groups. Such 'intervention-generated inequalities' (IGIs) may arise at a number of points in the implementation of an intervention, including intervention efficacy, service provision or access, uptake, and compliance.[3 4] Conversely, some interventions may reduce inequalities, if they are of greater benefit to disadvantaged groups.

A number of intervention types have been investigated for the possibility of IGIs, and there is a substantial body of theoretical work and guidance on the kinds of interventions which are likely to reduce or increase inequalities.[5–7] However, few studies have sought to bring together what is known about IGIs across the whole field of public health interventions. The aim of this paper is to provide an overview of evidence from systematic reviews in order to provide preliminary indications as to which types of interventions are more likely to produce IGIs, and which have the potential to reduce inequalities.

METHODS

The method used was a systematic review of reviews, with limited searching but systematic screening. Analogous methods have been widely used to produce 'rapid reviews': they aim to minimise selection bias, as in a full systematic review, but not to be fully comprehensive, and so cannot rule out publication bias.[8 9] We searched MEDLINE using the string "(inequalit$ or equit$ or inequit$ or disparit$).tw." in conjunction with SIGN's filter for systematic reviews (http://www.sign.ac.uk/methodology/filters.html#systematic), and searched the bibliography from a recent review of reviews on inequalities.[10] We included systematic reviews which evaluated the effectiveness of any non-healthcare intervention in a high-income country on any health outcome, and which reported differences in intervention effectiveness between population groups, defined in terms of the PROGRESS-Plus framework.[11 12] Full methods and the flow of literature are presented in the web-only appendix to this paper.

It should be noted that a differential intervention effect does not necessarily imply an IGI. Strictly speaking, we can identify an IGI only where we know that an intervention has created a health inequality where none existed at baseline, or widened an existing inequality. To confirm this, we would need detailed information on the study sample at baseline, showing that more disadvantaged groups on the relevant demographic (PROGRESS-Plus) dimension are worse off, or at least no better off, with respect to the relevant health variable(s). However, such baseline data are often not recoverable from secondary or tertiary research findings. For this reason, our analysis focuses on differences in intervention effect between groups of lower and higher socioeconomic status (SES; broadly defined to include measures such as income, occupational status, employment status, housing tenure or level of education), rather than on other PROGRESS-Plus factors, such as gender or ethnicity. This is because, while the existence and direction of an inequality gradient by SES for most health behaviour and health status variables is reasonably well established, and can in

What types of interventions generate inequalities? Evidence from systematic reviews

Theo Lorenc,[1] Mark Petticrew,[1] Vivian Welch,[2] Peter Tugwell[2]

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[1]Department of Social & Environmental Health Research,

ABSTRACT

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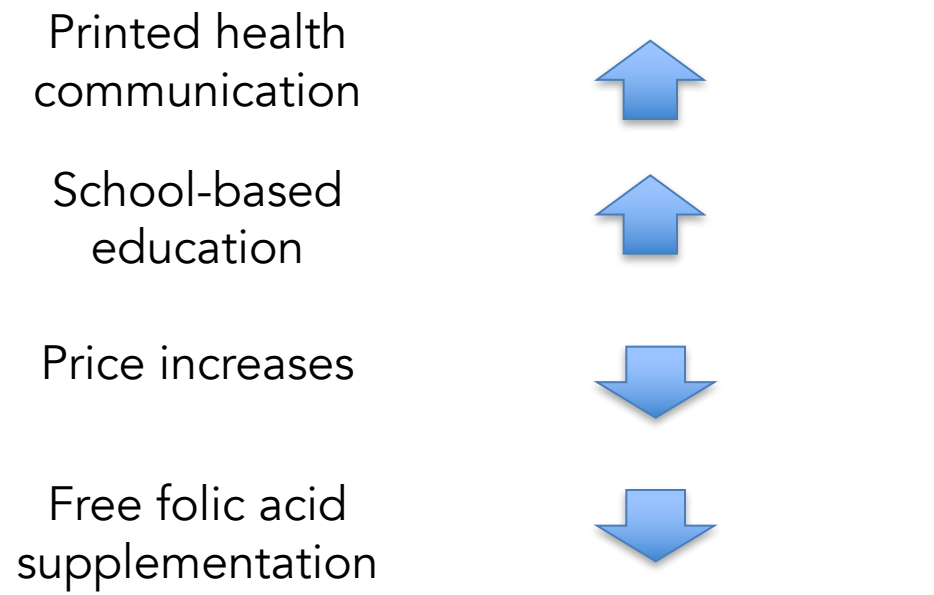

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A number of intervention types have been investigated for the possibility of IGIs, and there is a substantial body of theoretical work and guidance on the kinds of interventions which are likely to reduce or increase inequalities.[5–7] However, few studies have sought to bring together what is known about IGIs across the whole field of public health interventions. The aim of this paper is to provide an overview of evidence from systematic reviews in order to provide preliminary indications as to which types of interventions are more likely

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d. Loss of confidence in what we find/promote

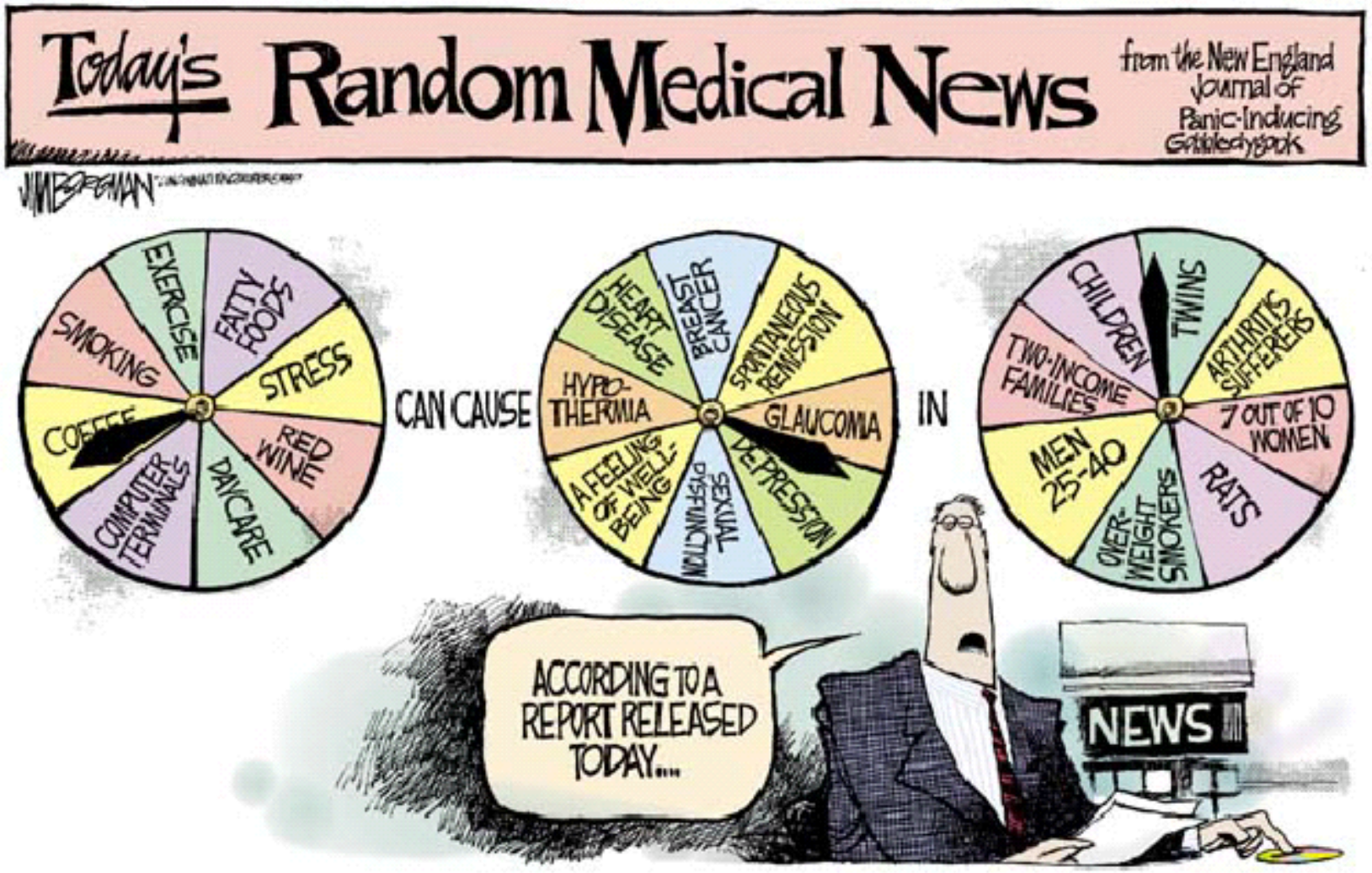
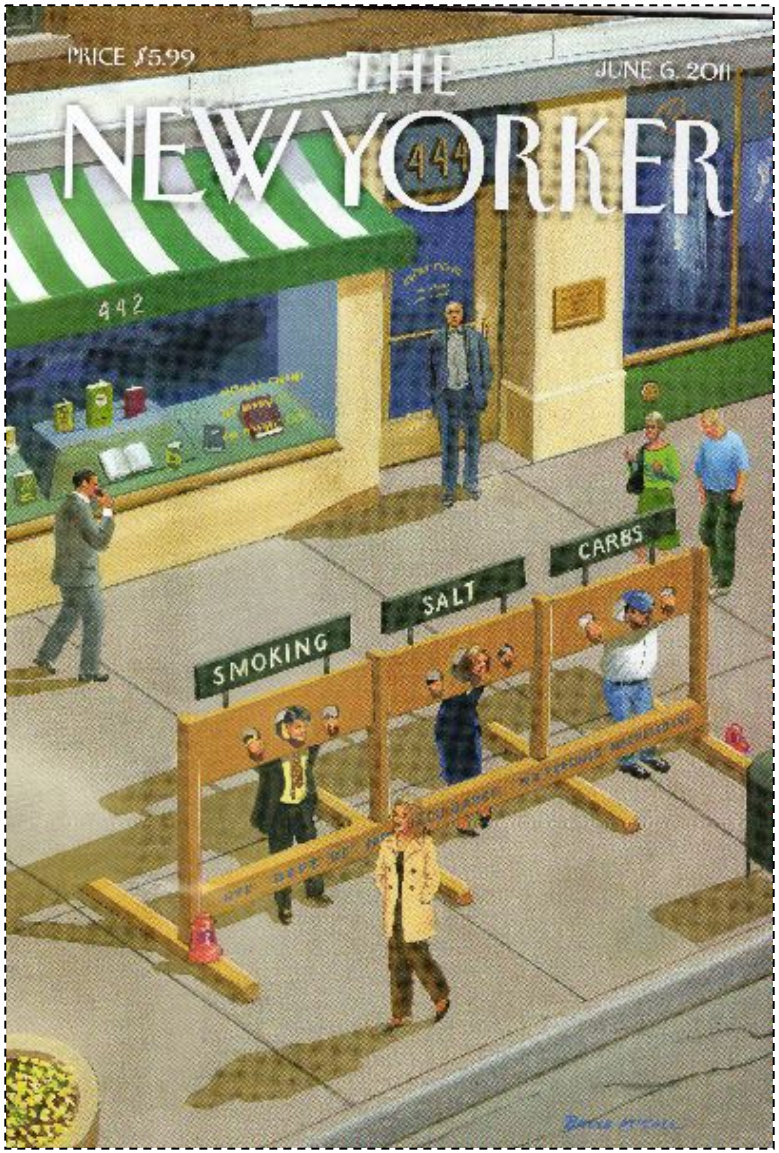


Figure 3: New England Journal of Panic-Inducing Gobbledygook.
Source: Jim Borgman, The Cincinnati Enquirer (27 April 1997, E4).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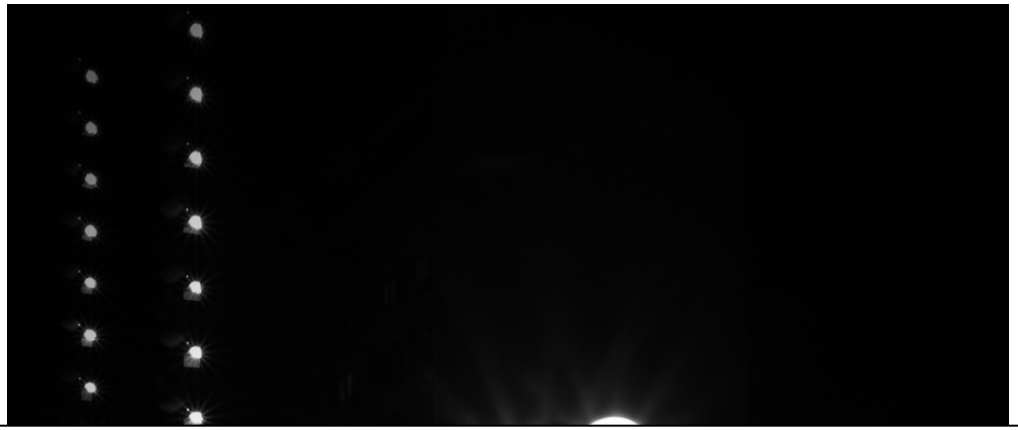
Anthony Taber. http://www.art.com/products/p15063130847-sa-i6842974/anthony-taber-square-peg-looking-at-round-hole-thinking-with-a-proper-diet-moderate-bu…-new-yorker-cartoon.htm

3. We know there are problems with this approach

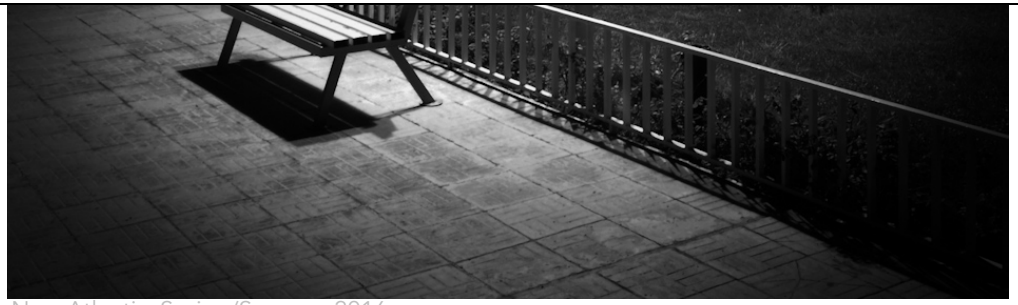
“We need to foster…research, not only by improving our methodology and sharing our scientific experience, but by helping to convince the...public and its legislators that prevention is far more important than treatment…and that the application of our findings to improve the health of the public must become the highest priority for health policy...”

Terris M. The Society for Epidemiologic Research (SER) and the future of epidemiology. American Journal of Epidemiology. 1992;136(8): 909-915.

http://36.media.tumblr.com/tumblr_lmampu8hpP1qffl4go1_1280.jpg



If mouse models are like looking for your keys under the street lamp, big data is like looking all over the world for your keys because you can — even if you don't know what they look like or where you might have dropped them or whether they actually fit your lock.



Sarewitz D. Saving science. The New Atlantis. Spring/Summer 2016

http://36.media.tumblr.com/tumblr_lmampu8hpP1qffl4go1_1280.jpg

“The.. dangerous threat to science comes from areas of research where the stakes are high but the validity of the science cannot be determined…In these cases, science delivers partial truths, any one of which can advance the career of a researcher and attract a constituency of believers among scientists, political interest groups, and members of the public alike.”

Sarewitz D. Saving science. The New Atlantis. Spring/Summer 2016

4. An alternative approach, a focus on consequences

Commentary

An Argument for a Consequentialist Epidemiology

Sandro Galea*

* Correspondence to Dr. Sandro Galea, Department of Epidemiology, Mailman School of Public Health, Columbia University, 722 W. 168th Street, Room 1508, New York, NY 10032-3727 (e-mail: sgalea@columbia.edu).

Initially submitted March 10, 2013; accepted for publication April 17, 2013.

"…academic epidemiology now spends most of its time concerned with identifying the causes and distributions of disease in human populations and far less of its time and imagination asking how we might improve population health, what might happen if a particular approach were taken to try to do so, where and when it may be appropriate to attempt inflections to the course of the health of populations, and whether our efforts to elucidate particular causes is usefully guiding our way to population health improvement."

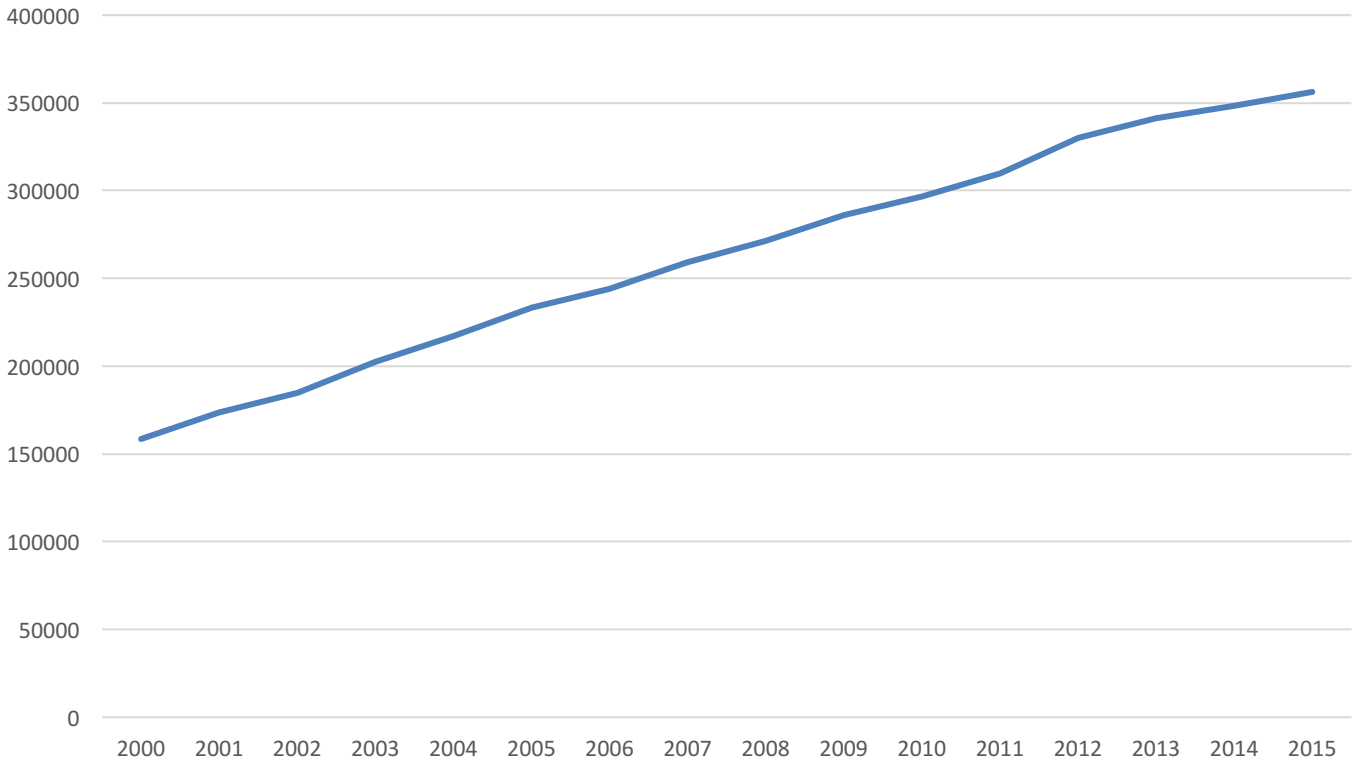
Epidemiology Matters

A new introduction to methodological foundations

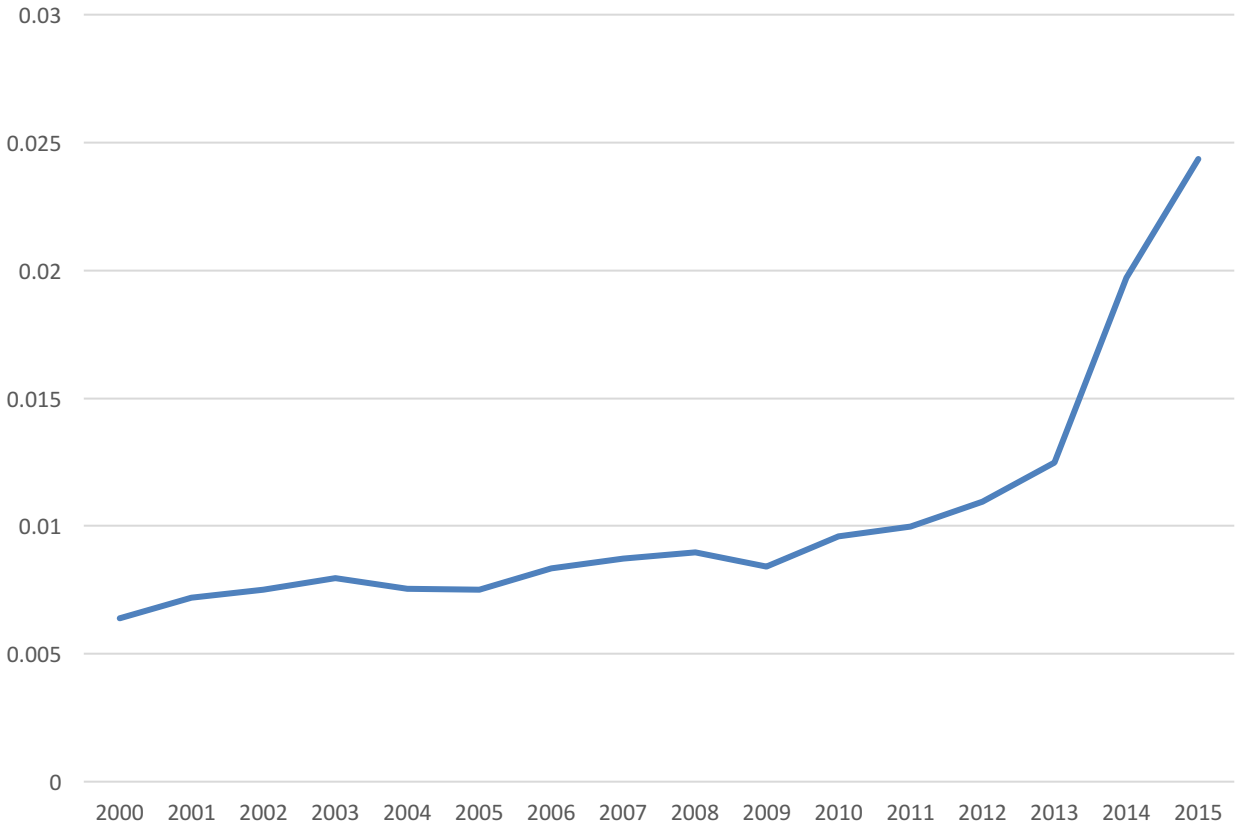
Katherine M. Keyes
Sandro Galea

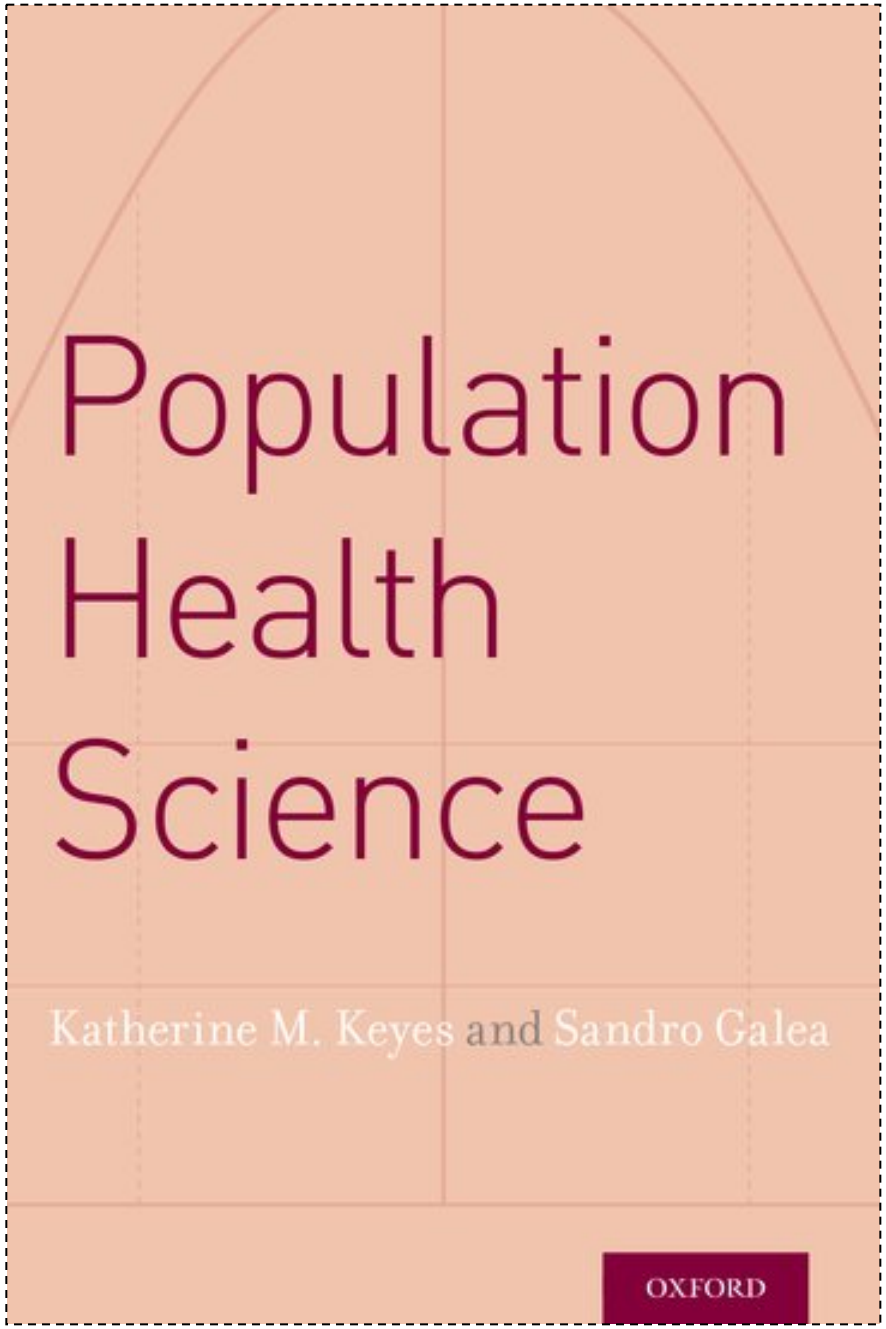
OXFORD

Public health, PubMed

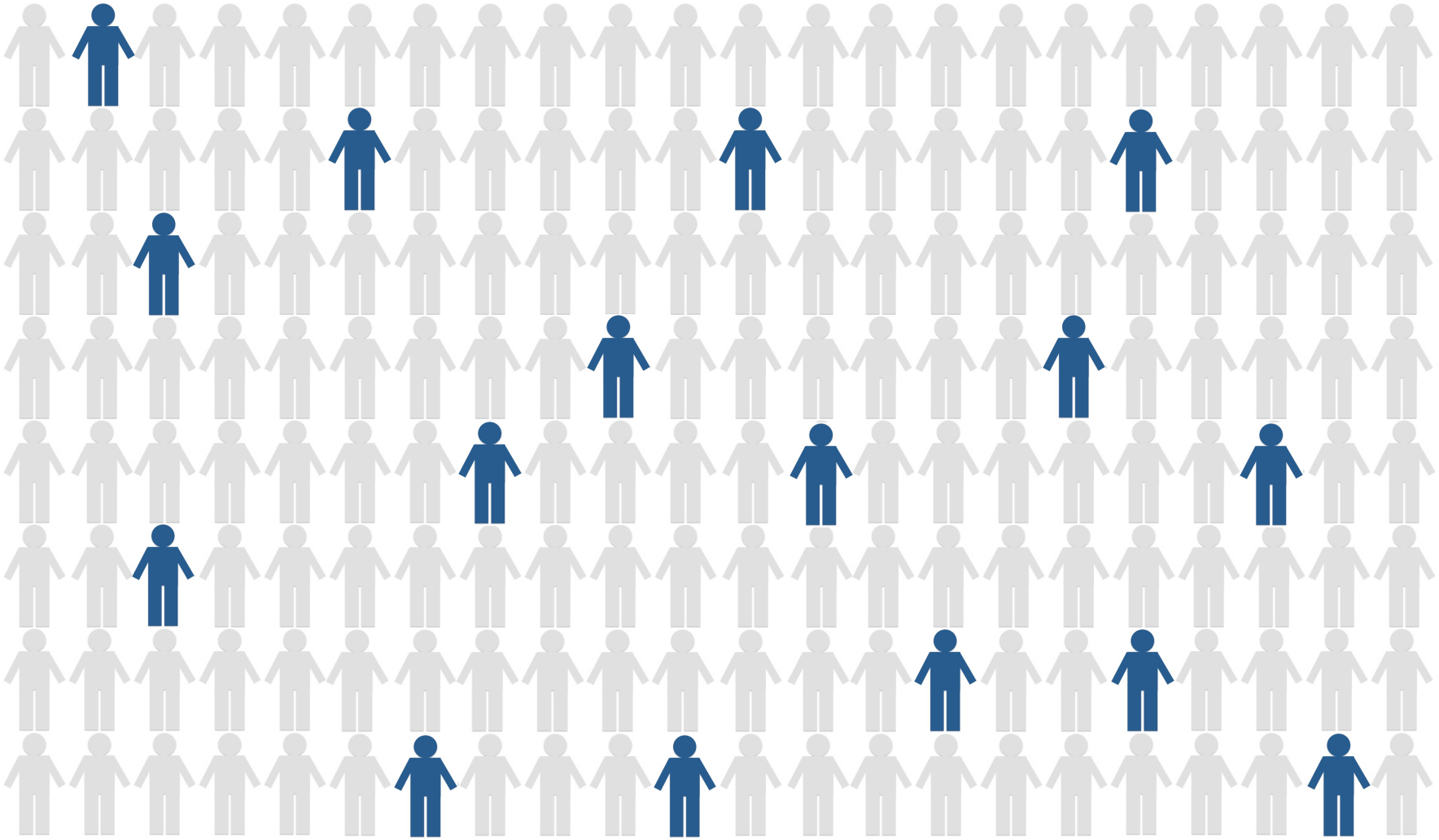


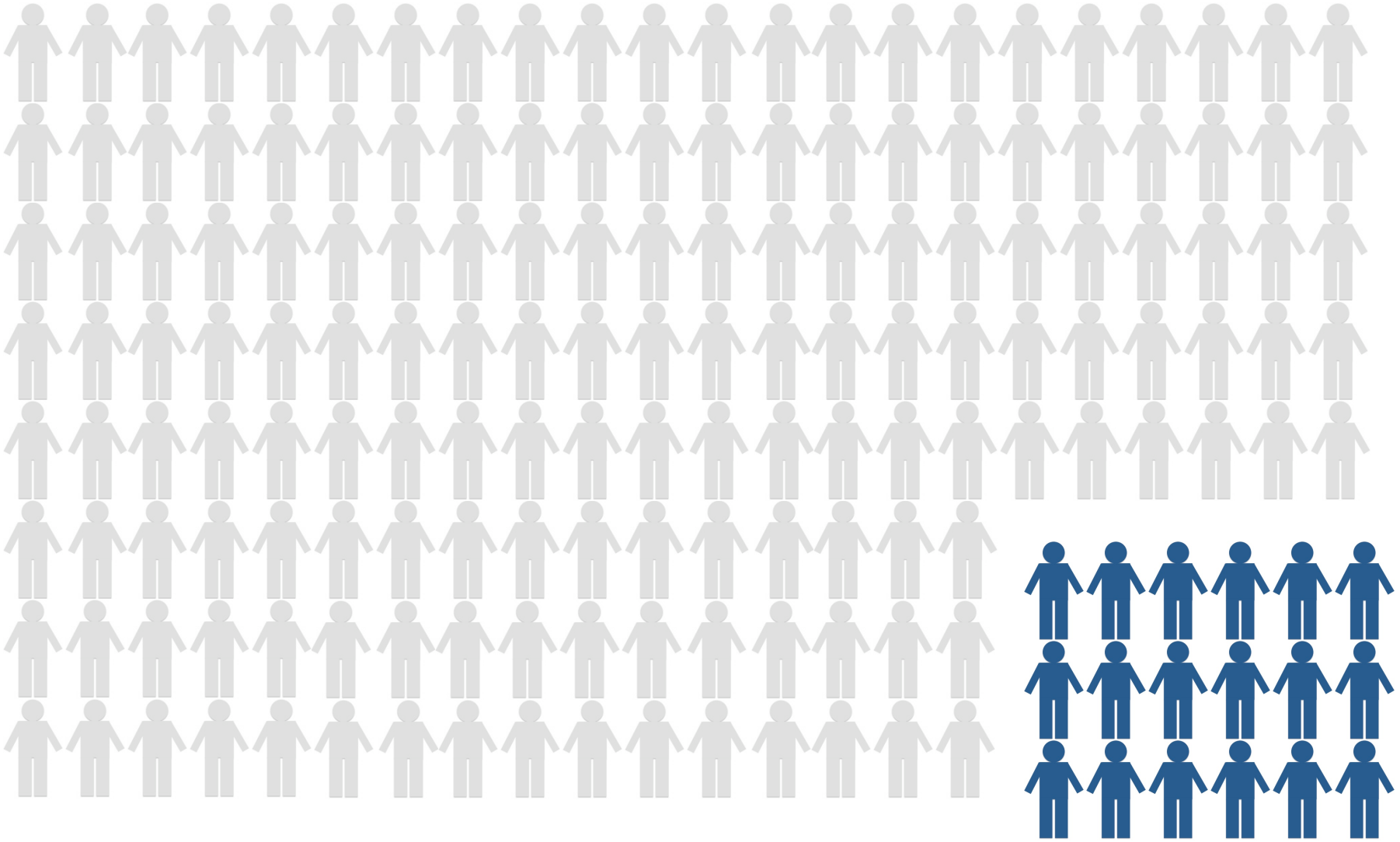
Population health science/public health, PubMed




Population
Health
Science
Katherine M. Keyes and Sandro Galea
OXFORD

http://graphics8.nytimes.com/images/2009/03/11/opinion/13population.480.jpg





What is population health science?

> Population health science is the study of the conditions that shape distributions of health within and across populations, and of the mechanisms through which these conditions manifest as the health of individuals

1. Population health manifests as a continuum.
2. The causes of differences in health across populations are not necessarily an aggregate of the causes of differences in health within populations.
3. Large benefits to population health may not improve the lives of all individuals.
4. The causes of population health are multilevel, accumulate throughout the life course, and are embedded in dynamic interpersonal relationships.
5. Small changes in ubiquitous causes may result in more substantial change in the health of populations than larger changes in rarer causes.
6. The magnitude of an effect of exposure on disease is dependent on the prevalence of the factors that interact with that exposure.
7. Prevention of disease often yields a greater return on investment than curing disease after it has started.
8. Efforts to improve overall population health may be a disadvantage to some groups; whether equity or efficiency is preferable is a matter of values.
9. We can predict health in populations with much more certainty than we can predict health in individuals.

Principle 1. Population health manifests as a continuum

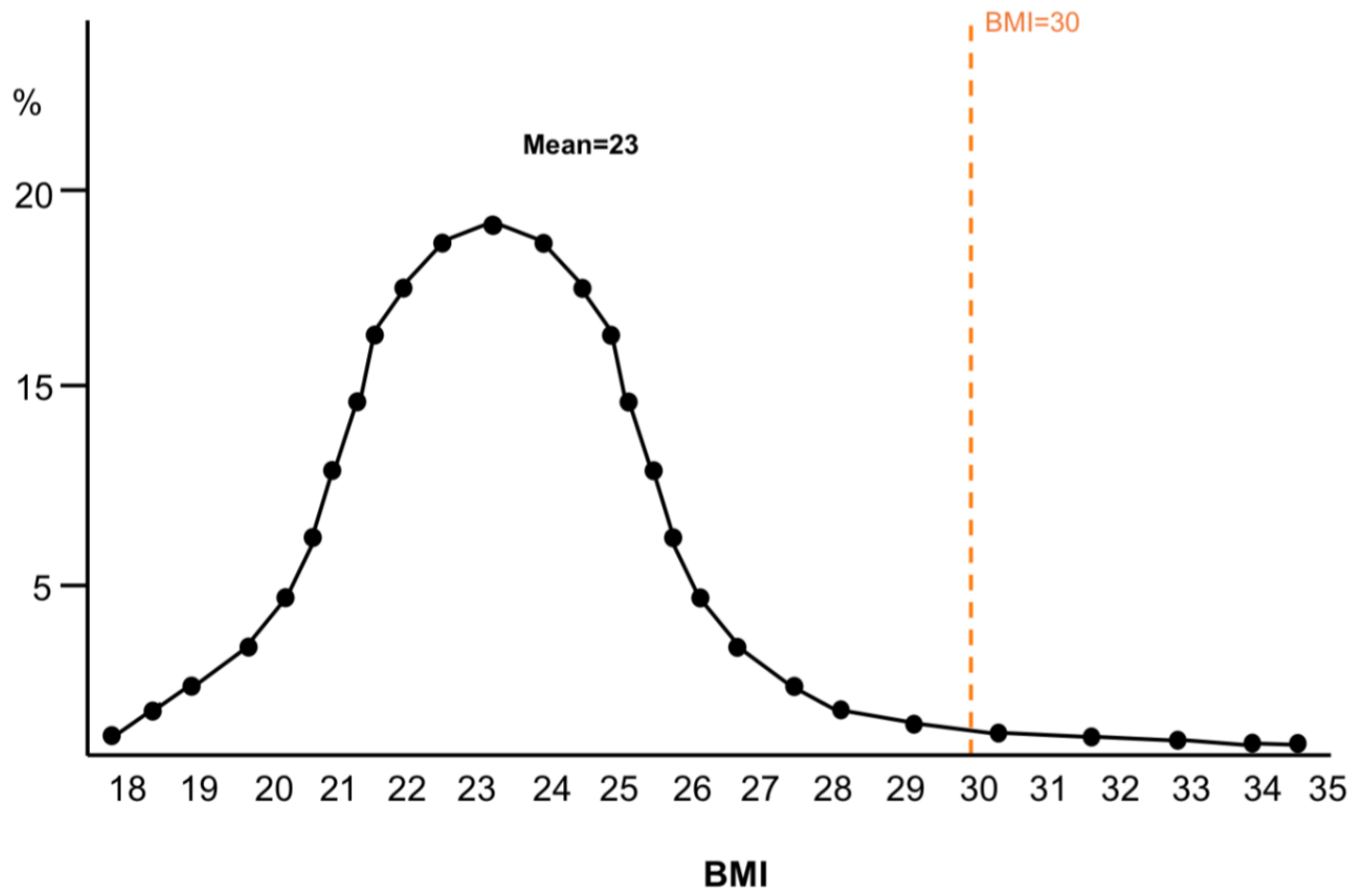
IL NOSTRO VENERDì

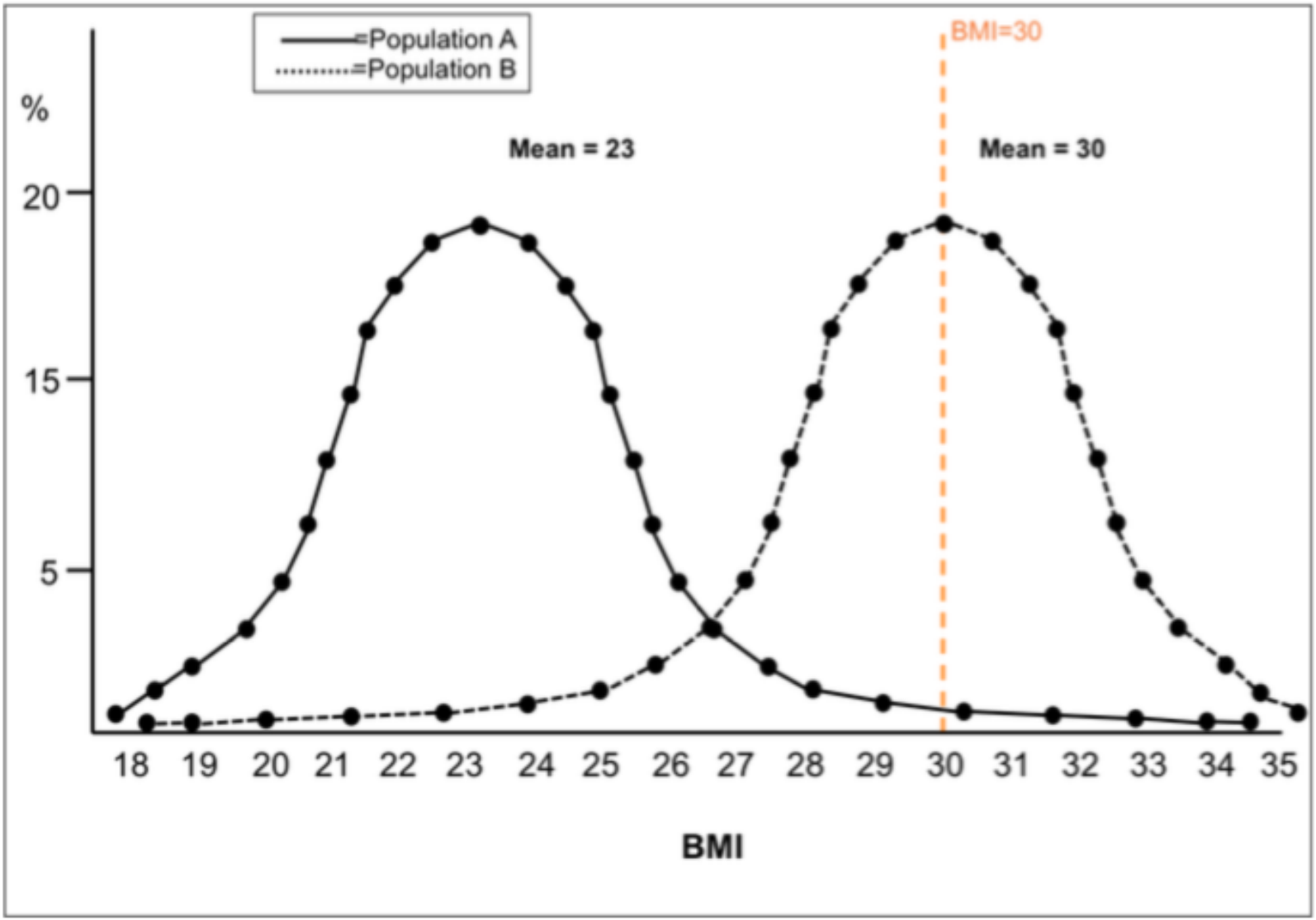
Culture

Divertente campagna per la prevenzione dell'obesità

Figure 1. Distribution of BMI in two populations illustrating health as a continuum in the population

Panel A




=Population A
=Population B
BMI=30
%
Mean = 23
Mean = 30
20
15
5
18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35
BMI

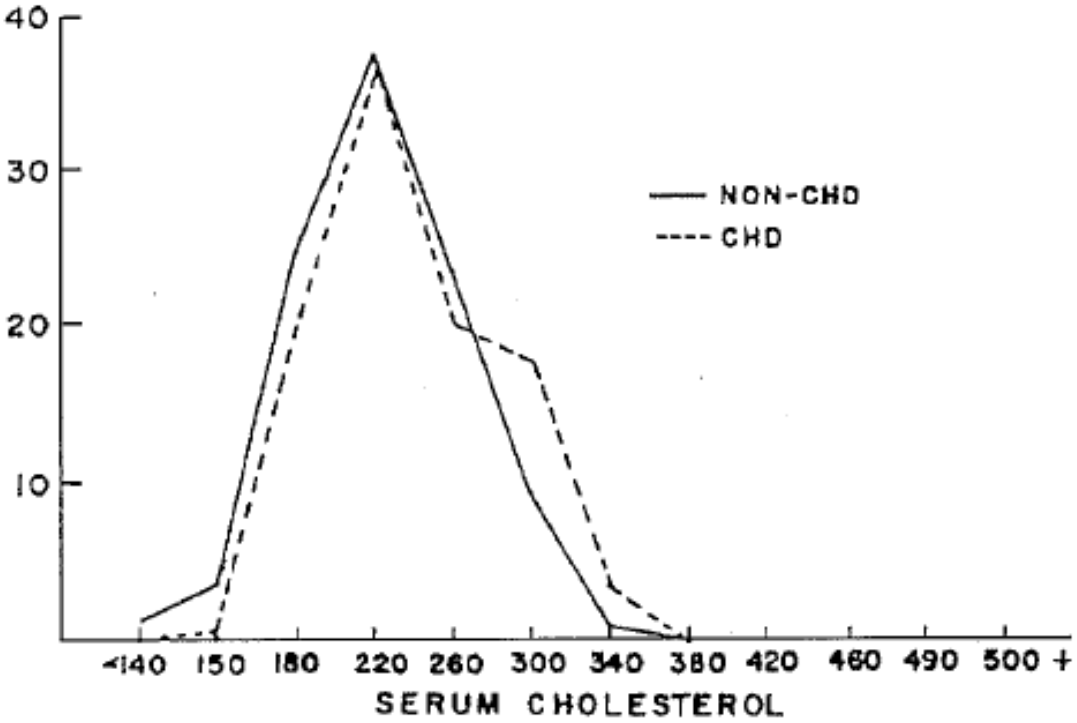
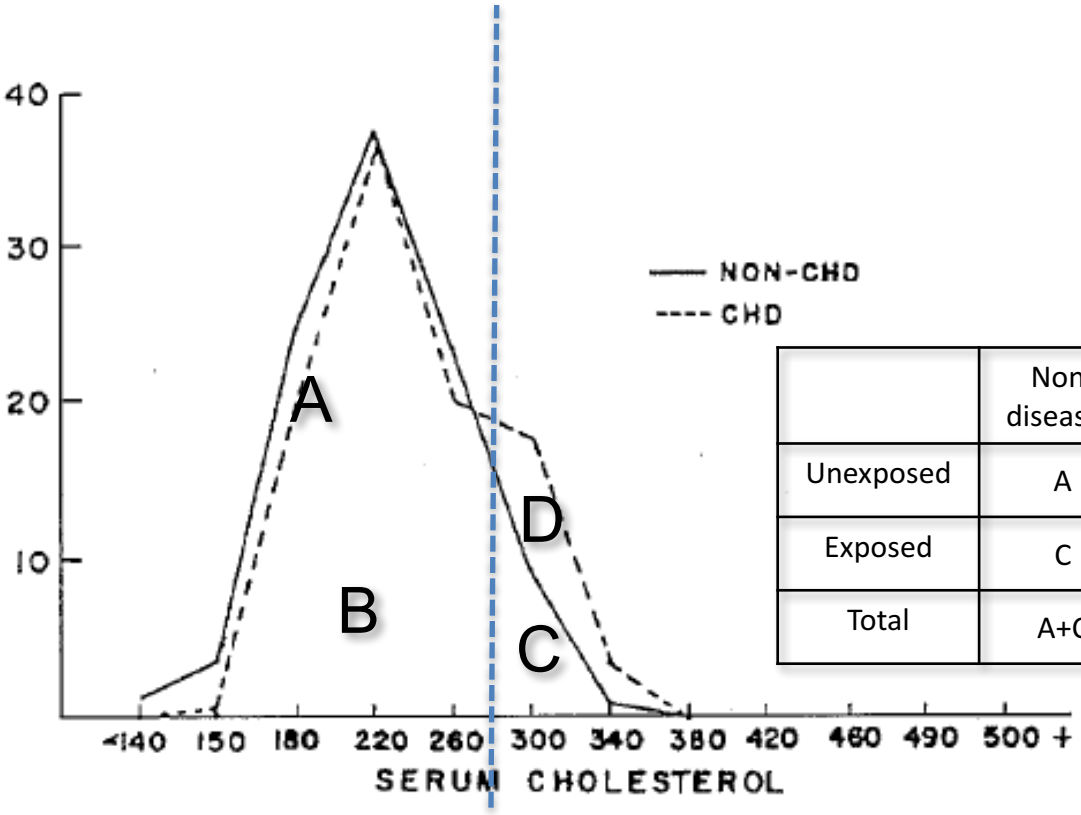


Figure 3 Percentage distribution of serum cholesterol levels (mg/dl) in men aged 50–62 who did or did not subsequently develop coronary heart disease (Framingham Study[5])



	Non-diseased	Diseased	Total
Unexposed	A	B	A+B
Exposed	C	D	C+D
Total	A+C	B+D	N

Figure 3 Percentage distribution of serum cholesterol levels (mg/dl) in men aged 50–62 who did or did not subsequently develop coronary heart disease (Framingham Study[5])

Rose G. Sick individuals and sick populations. International Journal of Epidemiology. 1985;14:32-38

Principle 2. The causes of differences in health across populations are not necessarily an aggregate of the causes of differences in health within populations

Figure 1. Conceptualizing causation at the individual level: 20 students in a classroom

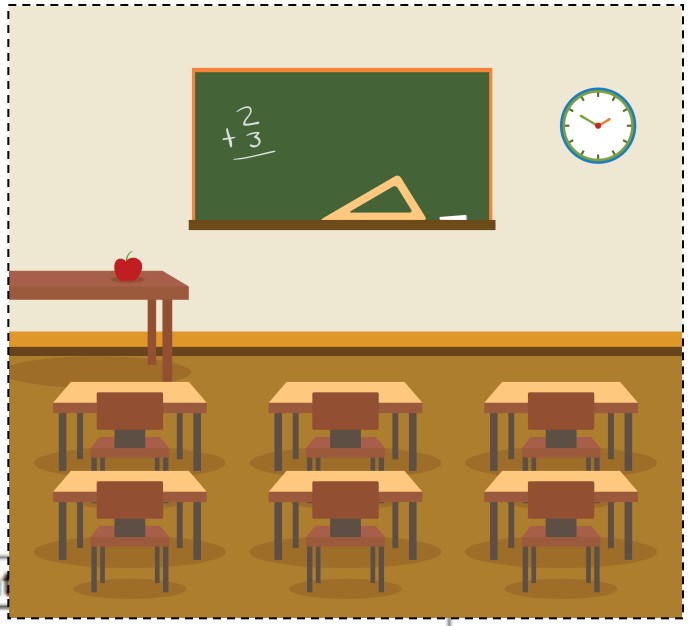


Figure 1. Conceptualizing causation at the individual level: 20 student

Why does Joe have asthma but not Leila?

Figure 2. Conceptualizing causation at the population level: comparing two classrooms

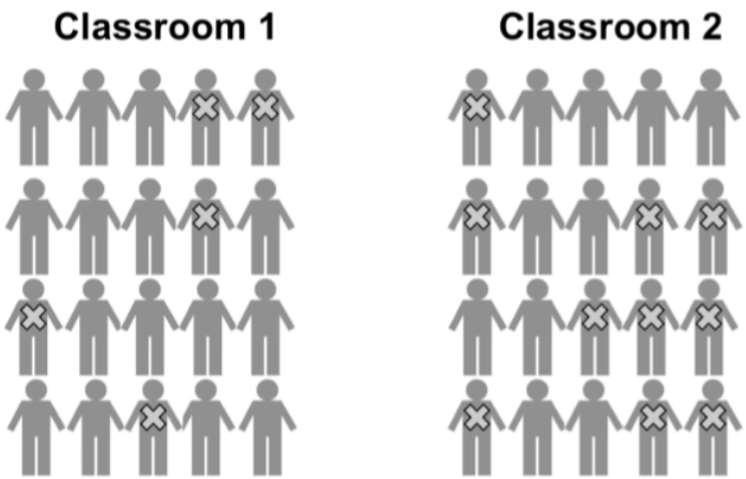
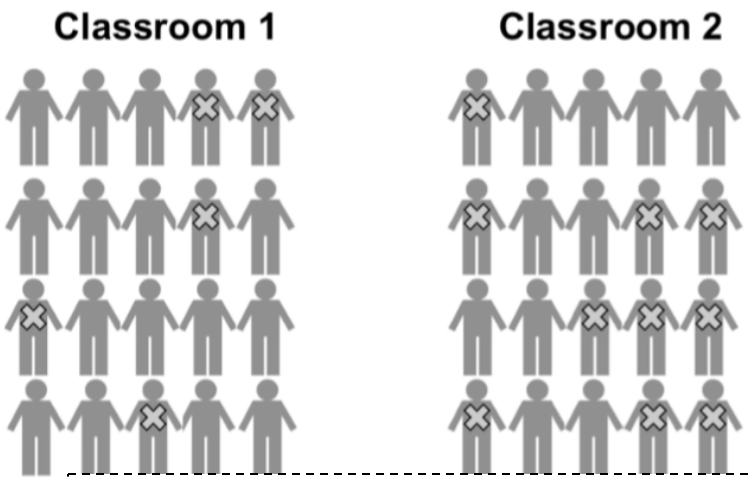


Figure 2. Conceptualizing causation at the population level: comparing two classrooms



Why there are twice as many cases of asthma in Classroom 2 compared with Classroom 1?

Principle 3. Large benefits to population health may not improve the lives of all individuals

What are the causes of high blood pressure among civil servants in London?

Figure 1. Hypothetical data on the systolic blood pressure of London civil servants

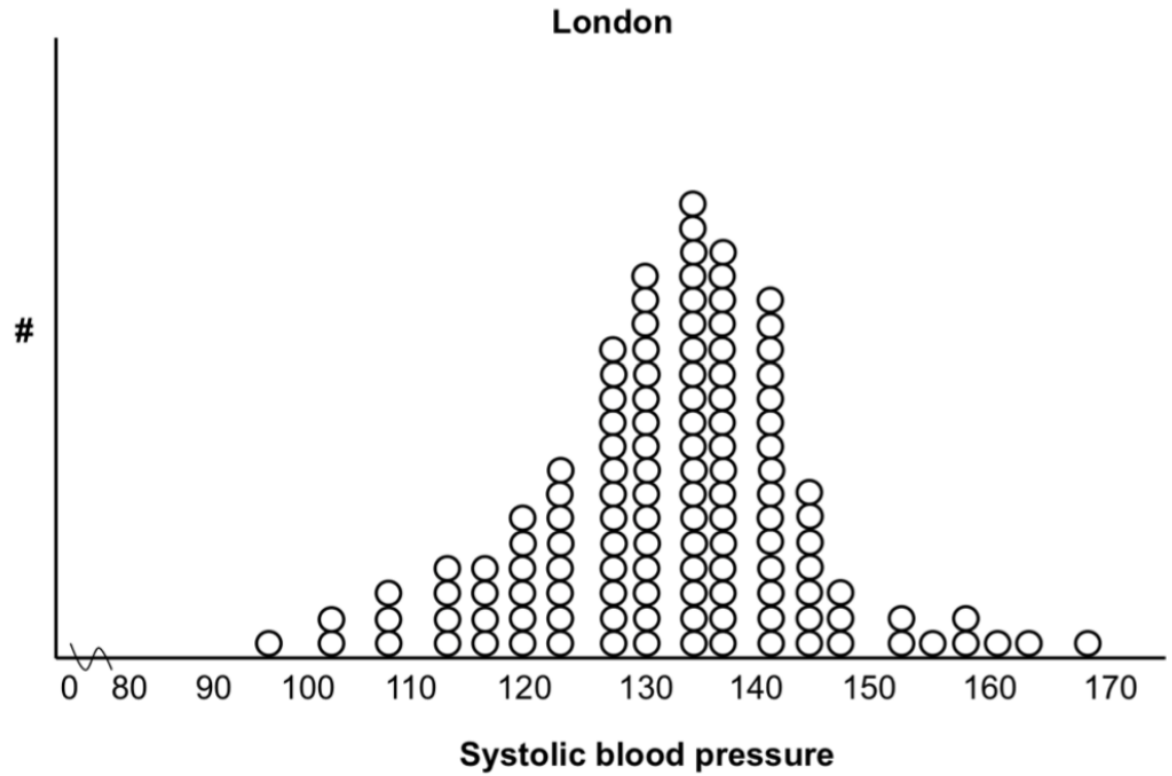
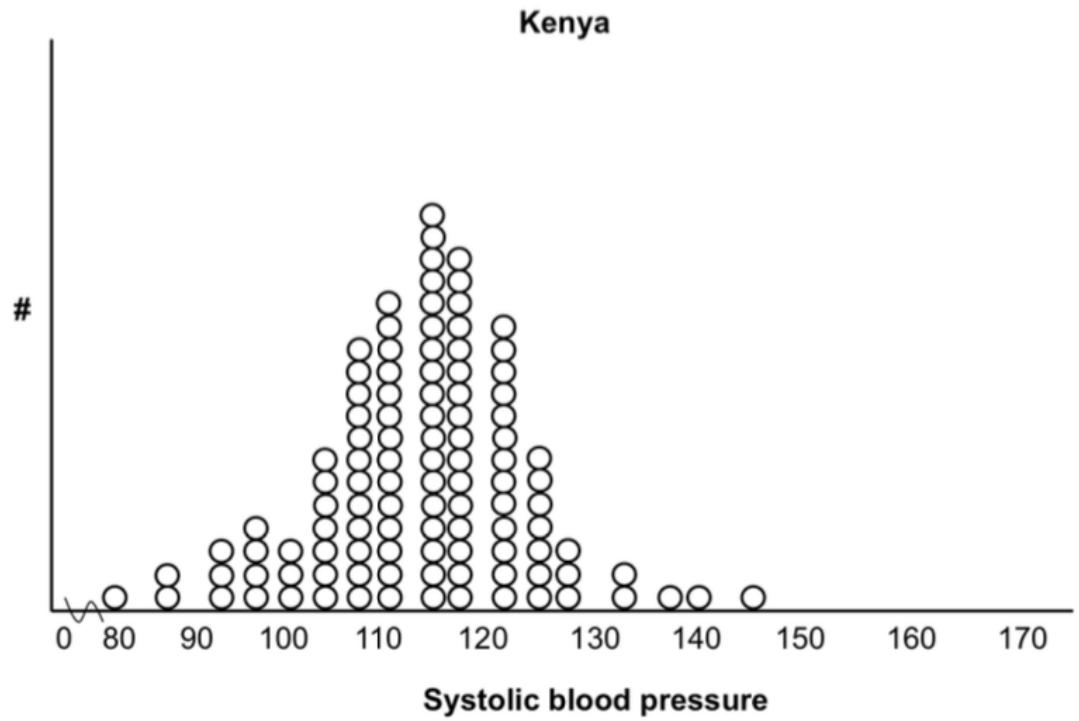
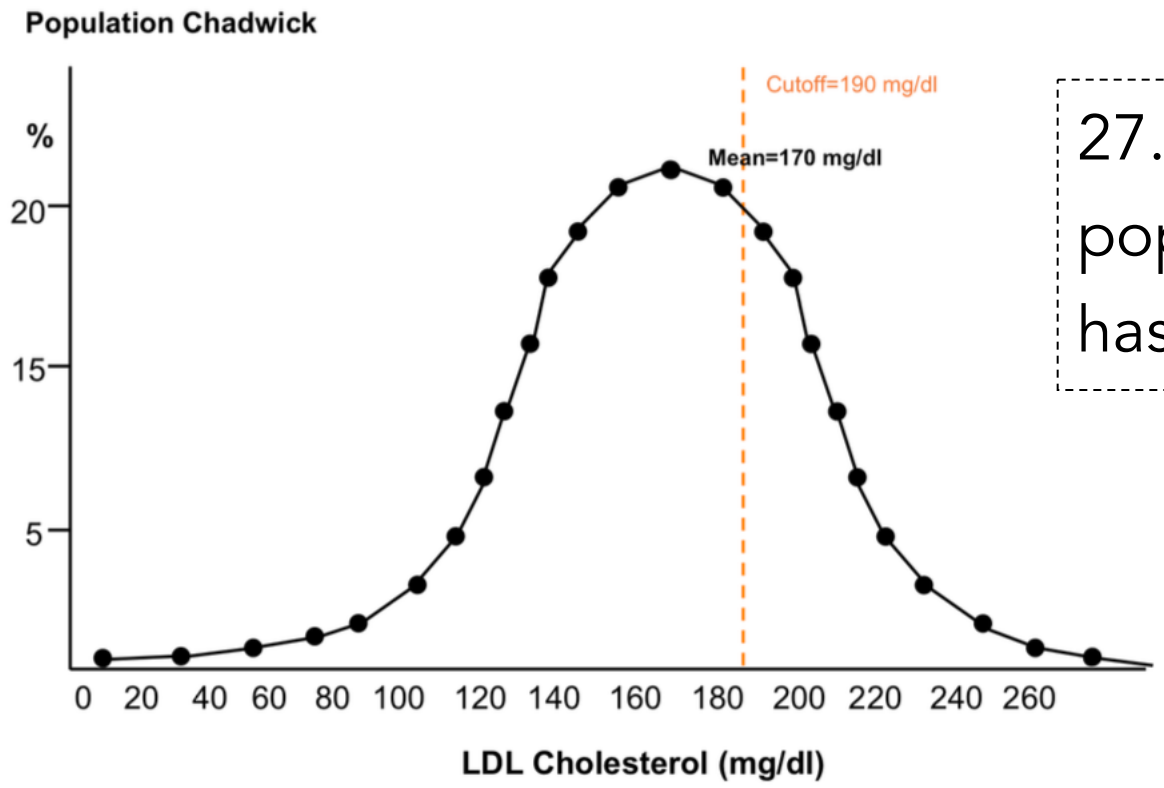


Figure 2: Hypothetical data on the systolic blood pressure of Kenyan nomads



How do we best lower LDL cholesterol in the towns of Chadwick and Farr?

Figure 3. Distribution of LDL cholesterol in Chadwick



27.5% of population has high LDL

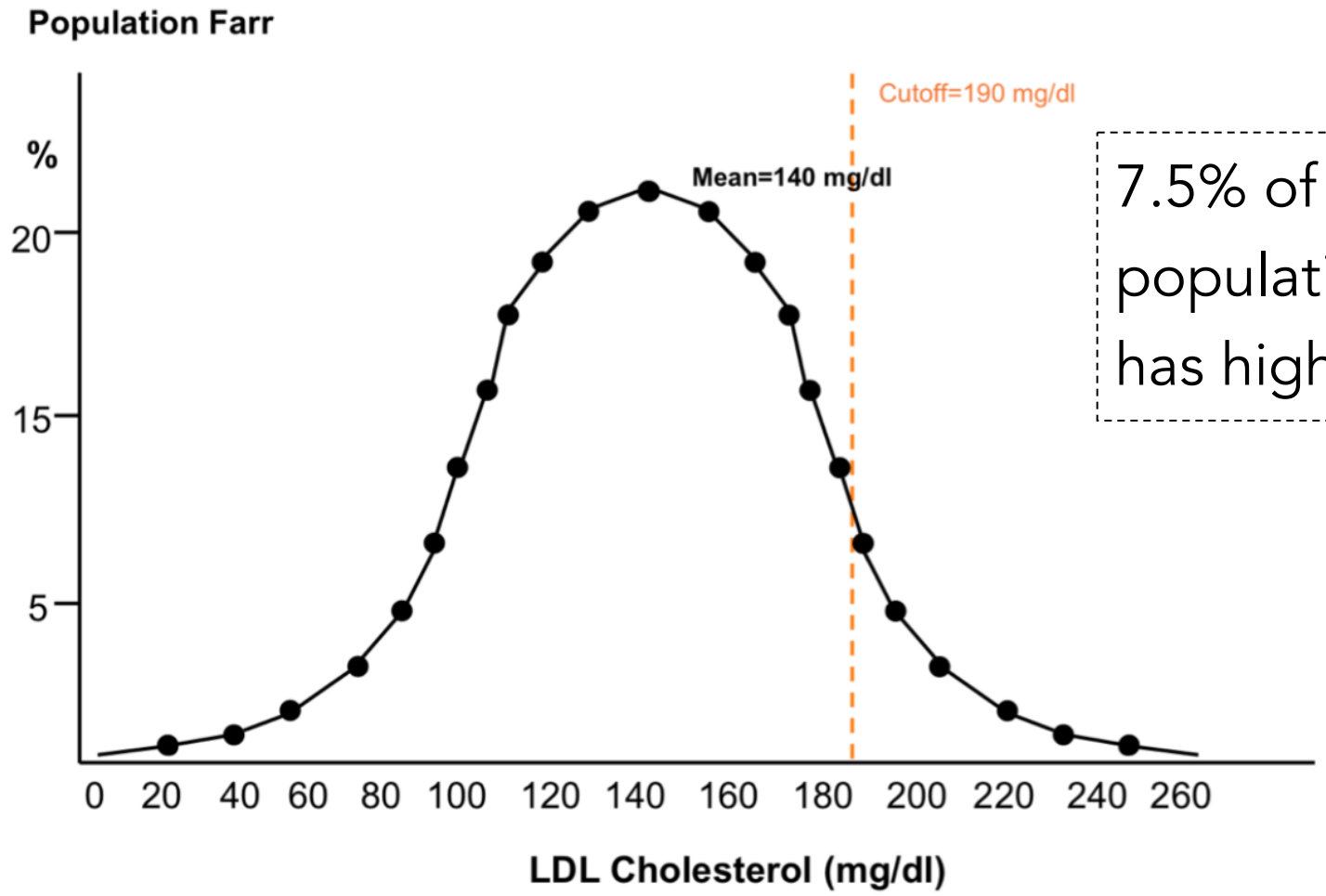
Table 1.The relation between potato chip eating and cholesterol in Chadwick

	High LDL cholesterol	Low LDL cholesterol	Total
Chip eater	150	350	500
Non-chip eater	125	375	500
Total	275	725	1000

$$\text{Risk difference} = \left(\frac{150}{500}\right) - \left(\frac{125}{500}\right) = 0.05$$

i.e., 5 additional cases of high LDL cholesterol for every 100 additional chip eaters

Figure 4. Distribution of LDL cholesterol in Farr



7.5% of population has high LDL

Table 2. The relation between potato chip eating and cholesterol in Farr

	High LDL cholesterol	Low LDL cholesterol	Total
Chip eater	50	450	500
Non-chip eater	25	475	500
Total	75	925	1000

$$\text{Risk difference} = \left(\frac{50}{500}\right) - \left(\frac{25}{500}\right) = 0.05$$

i.e., also 5 additional cases of high LDL cholesterol for every 100 additional chip eaters

In both populations the risk of high LDL linked to potato chip eating is the same. But, in Population Chadwick, 27.5% of the population has high LDL cholesterol, compared with 7.5% of the population in Population Farr. What is driving the difference?

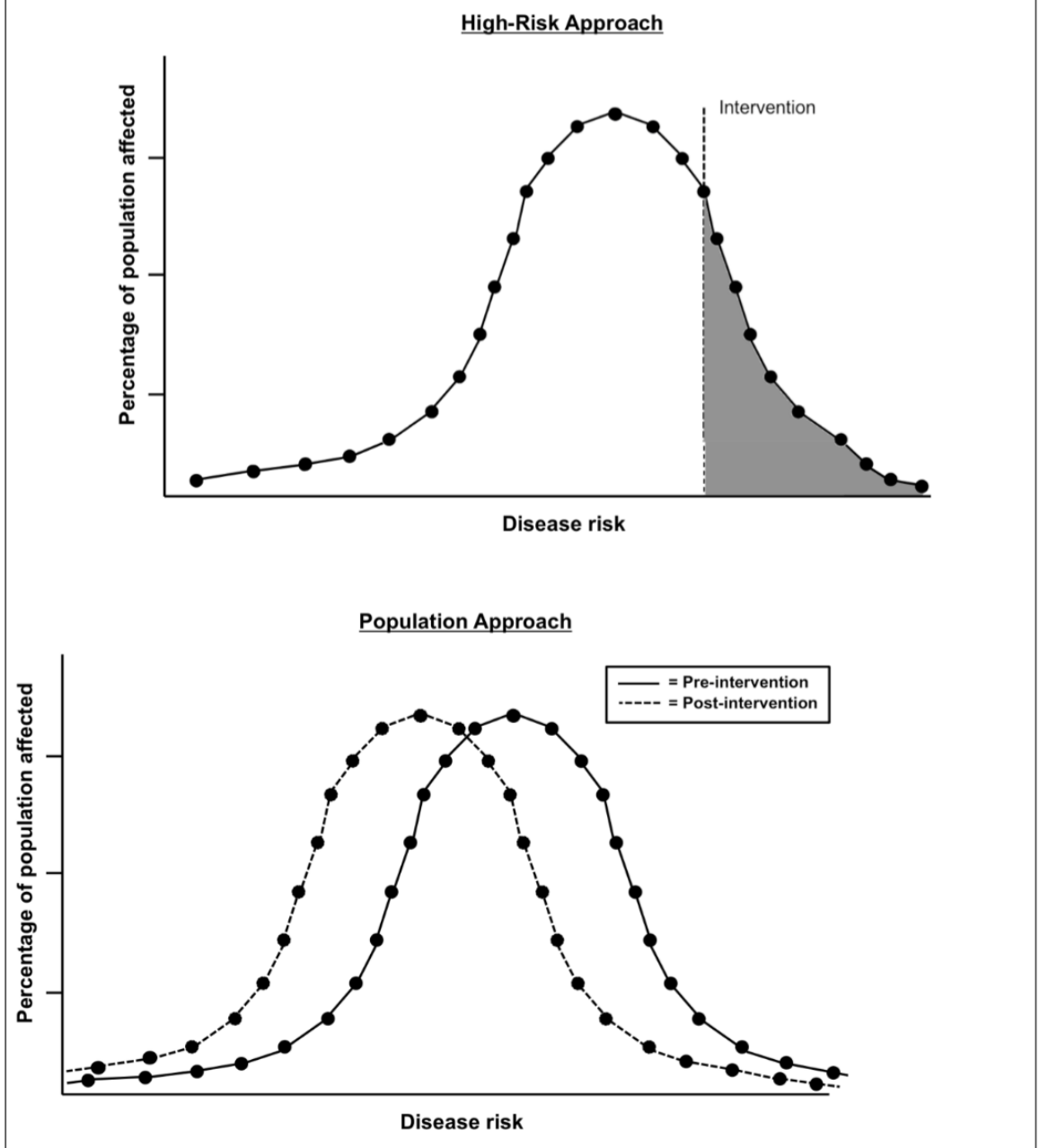
Table 3. The relation between childhood poverty and cholesterol in both Chadwick (all exposed to childhood poverty) and Farr (all unexposed to childhood poverty)

	High LDL cholesterol	Low LDL cholesterol	Total
Childhood poverty	275	725	1000
No poverty	75	925	1000
Total	350	1650	2000

$$\text{Risk difference} = \left(\frac{275}{1000}\right) - \left(\frac{75}{1000}\right) = 0.20$$

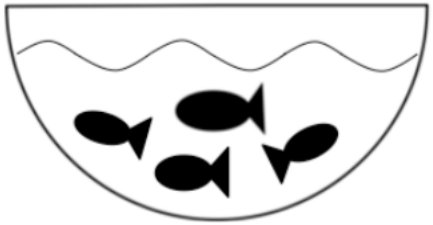
i.e., there is an excess of 20 cases of high LDL cholesterol for every 100 adults exposed to childhood poverty across these populations

Figure 7. High-risk and population approaches to disease prevention

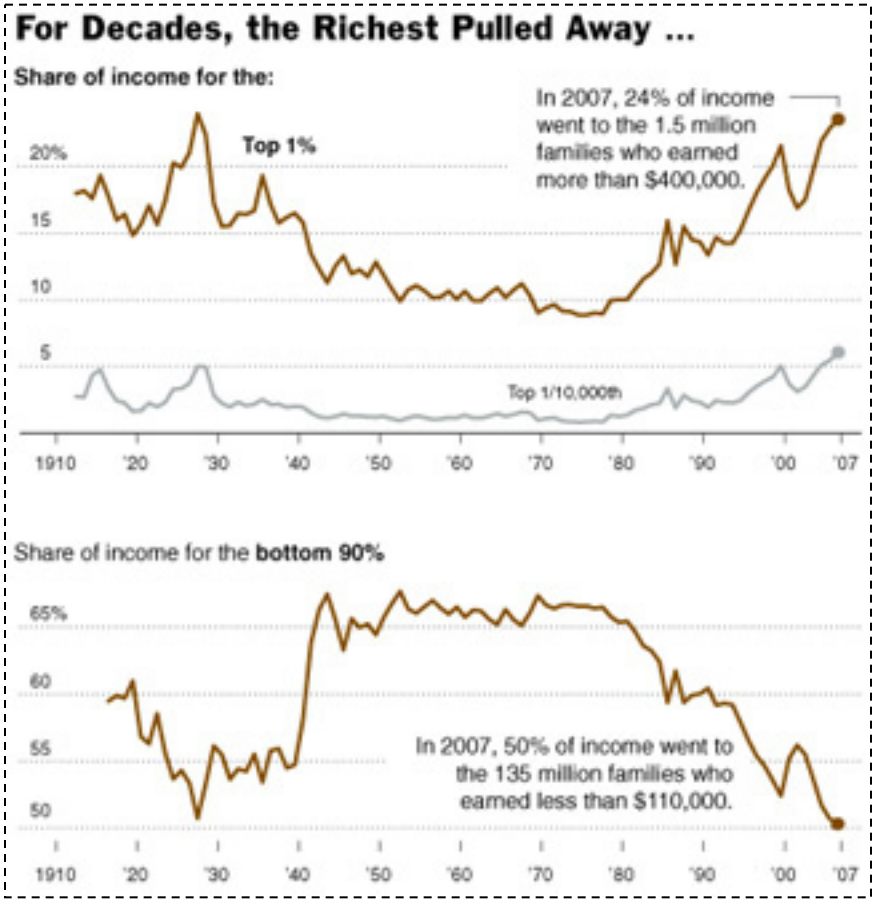


Principle 5. Small changes in ubiquitous causes may result in more substantial change in the health of populations than larger changes in rarer causes

Figure 1. A metaphor for ubiquity



The goldfish are surrounded by water and everything they do is influenced by the quality of the water in which they live; therefore, water is a ubiquitous factor influencing the fish and needs to be taken into consideration every time we may want to improve the lives of the fish.



http://farm3.static.flickr.com/2499/4056337199_a4620eb2ea.jpg

Table 1. Five-year mortality for those 18-65 in country with high income inequality

	# died	# alive	total
IV drug user	50	950	1000
Non-IV drug user	400	49600	50000
Total	450	50550	51000

$$RR = \frac{\frac{50}{1000}}{\frac{400}{50{,}000}} = 6.25$$

$$RD = \left(\frac{50}{1000}\right) - \left(\frac{400}{50{,}000}\right) = 0.042$$

The prevalence of IV drug use is 1.96% and IV drug users have 6.25 times the risk of mortality compared with non-IV drug users. For every 100 IV drug users, we would expect 4.2 additional deaths.

Suppose we reduce income inequality by 25%, keeping prevalence of IV drug use the same, but reducing excess mortality in all groups

Table 2. Five-year mortality for those 18-65 after reduction in income inequality

	# died	# alive	total
IV drug user	40	960	1000
Non-IV drug user	320	49680	50000
	360	50640	51000

Table 3. Comparison of 5-year mortality for those 18-65 before and after change in income inequality

	# died	# alive	Total
Before change in income inequality	450	50550	51000
After change in income inequality	360	50640	51000

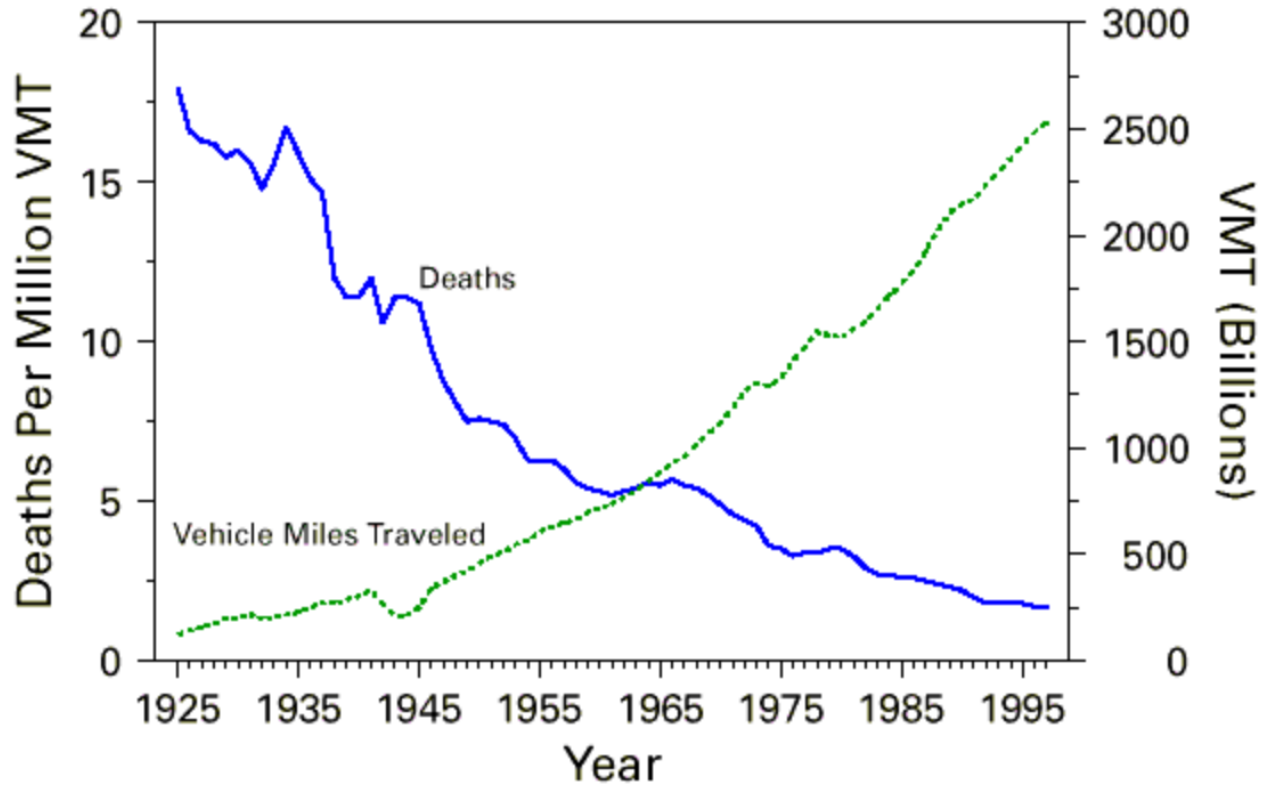
$$RR = \frac{\frac{450}{51000}}{\frac{360}{51000}} = 1.25$$

$$RD = \left(\frac{450}{51000}\right) - \left(\frac{360}{51000}\right) = 0.0018$$

Therefore, those in the unequal society had 1.25 times the risk of death, and we would expect 1.8 deaths per 1,000 persons exposed to the unequal society

In the hypothetical income inequality intervention, we have “saved” 90 lives (450-360=90). In our IV drug use example, we found that we would “save” an additional 4.2 times as many lives per 100 users; with 1000 total users in our population, we would estimate that we save a maximum of 42 lives even if all IV drug users stopped using.

FIGURE 1. Motor-vehicle–related deaths per million vehicle miles traveled (VMT) and annual VMT, by year — United States, 1925–1997



http://www.cdc.gov/mmwr/preview/mmwrhtml/mm4818a1.htm

Crack Babies: The Worst Threat Is Mom Herself

By Douglas J. Besharov

LAST WEEK in this city, Greater Southeast Community Hospital released a 7-week-old baby to her homeless, drug-addicted mother even though the child was at severe risk of pulmonary arrest. The hospital's explanation: "Because [the mother] demanded that the baby be released."

The hospital provided the mother with an apnea monitor to warn her if the baby stopped breathing while asleep, and trained her in CPR. But on the very first night, the mother went out drinking and left the child at a friend's house—without the monitor. Within seven hours, the baby was dead. Like Dooney Waters, the 6-year-old living in his mother's drug den, whose shocking story was reported in The Washington Post last week, this child was all but abandoned by ities.

September 17, 1989

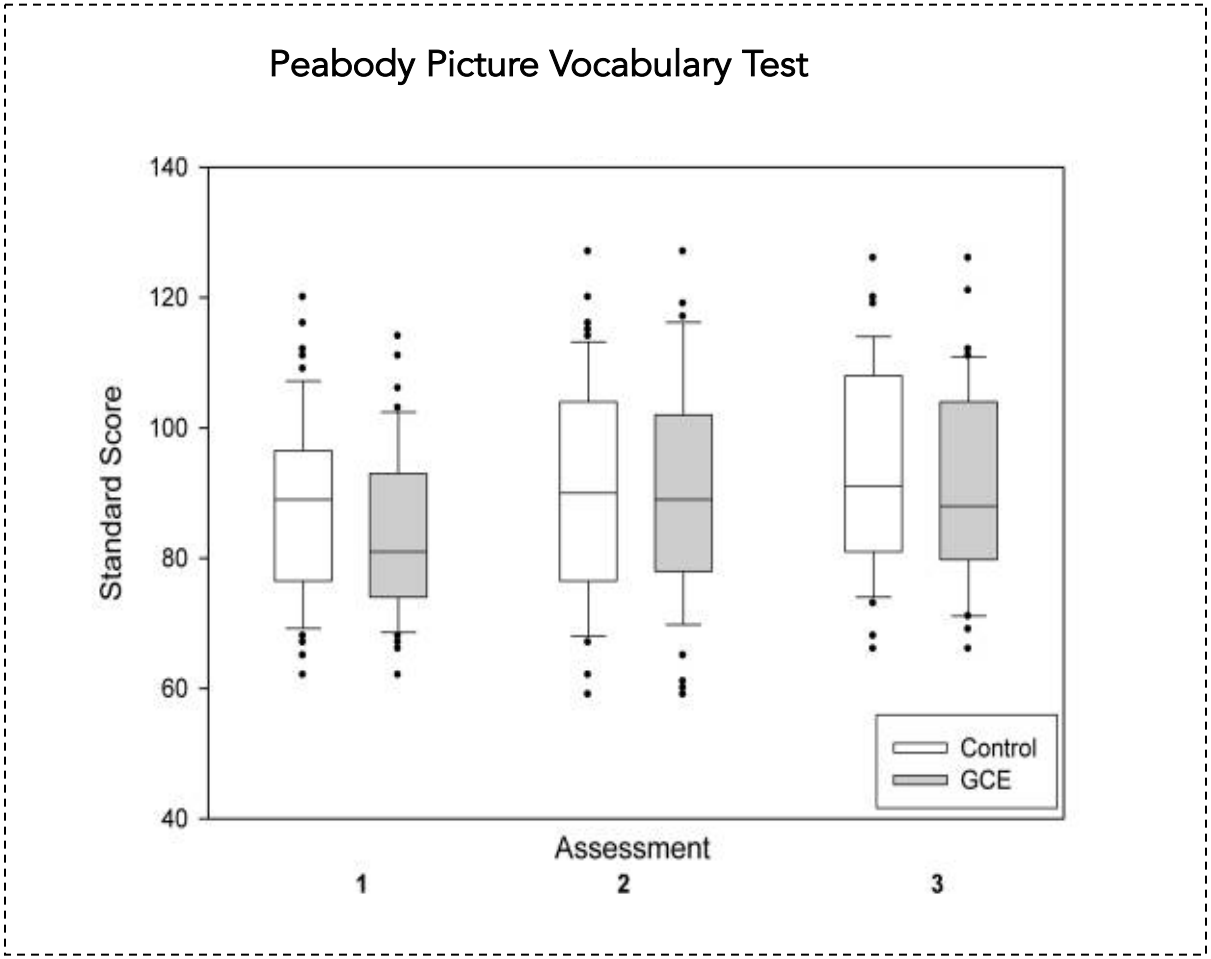
Crack's Toll Among Babies: A Joyless View

September 6, 1988

Cocaine: Litany of Fetal Risks Grows

CHILDREN OF COCAINE

(By Charles Krauthammer)



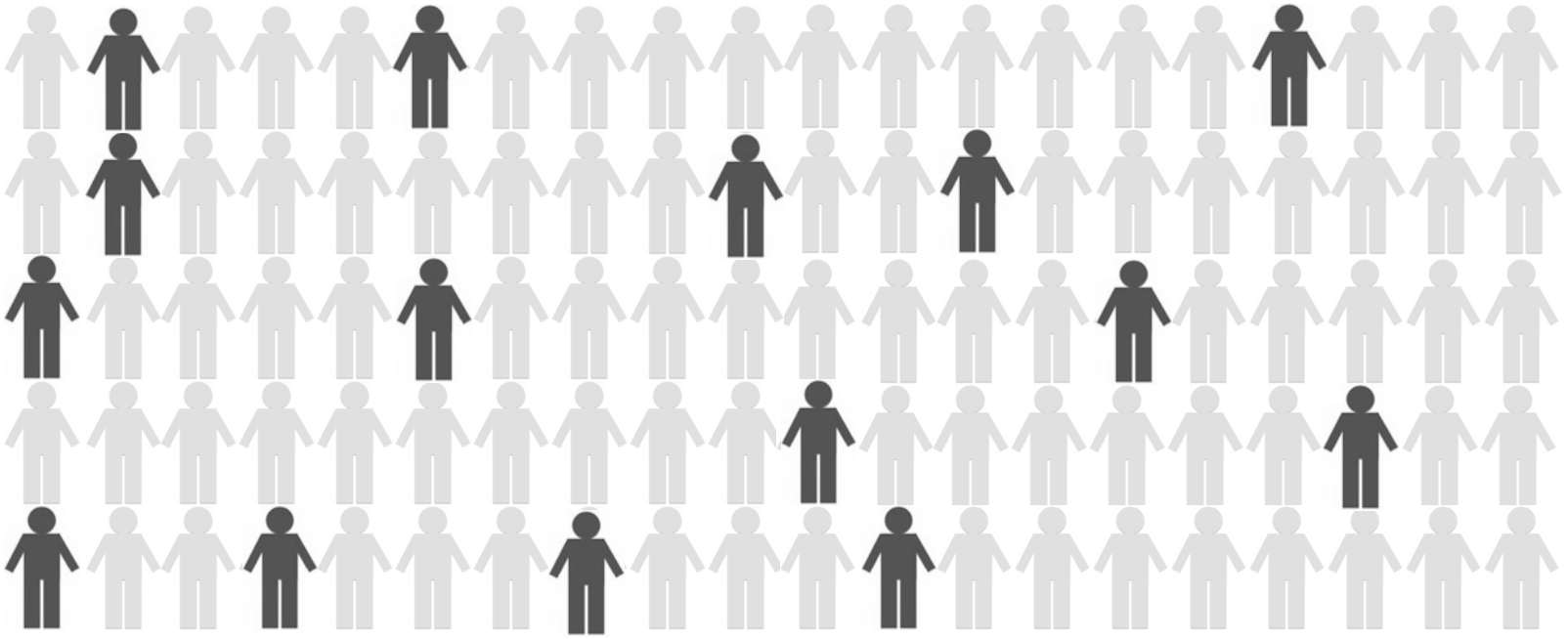
Betancourt LM et al. Adolescents with and without gestational cocaine exposure: Longitudinal analysis of inhibitory control, memory and receptive language. *Neurotoxicol Teratol* 2011; 33(1): 36-46.

Predictor for Peabody Picture Vocabulary Test score	Coefficient	P-value
Gestational cocaine exposure	-2.89	0.26
Assessment no.	2.72	<0.001
Gestational cocaine exposure x assessment no.	0.58	0.51
Age at 1st assessment	-0.36	0.76
Female gender	-4.93	0.058
Parental nurturance	-0.31	0.89
Environmental stimulation	5.91	0.039
Caregiver BDI-II depression score	0.03	0.84

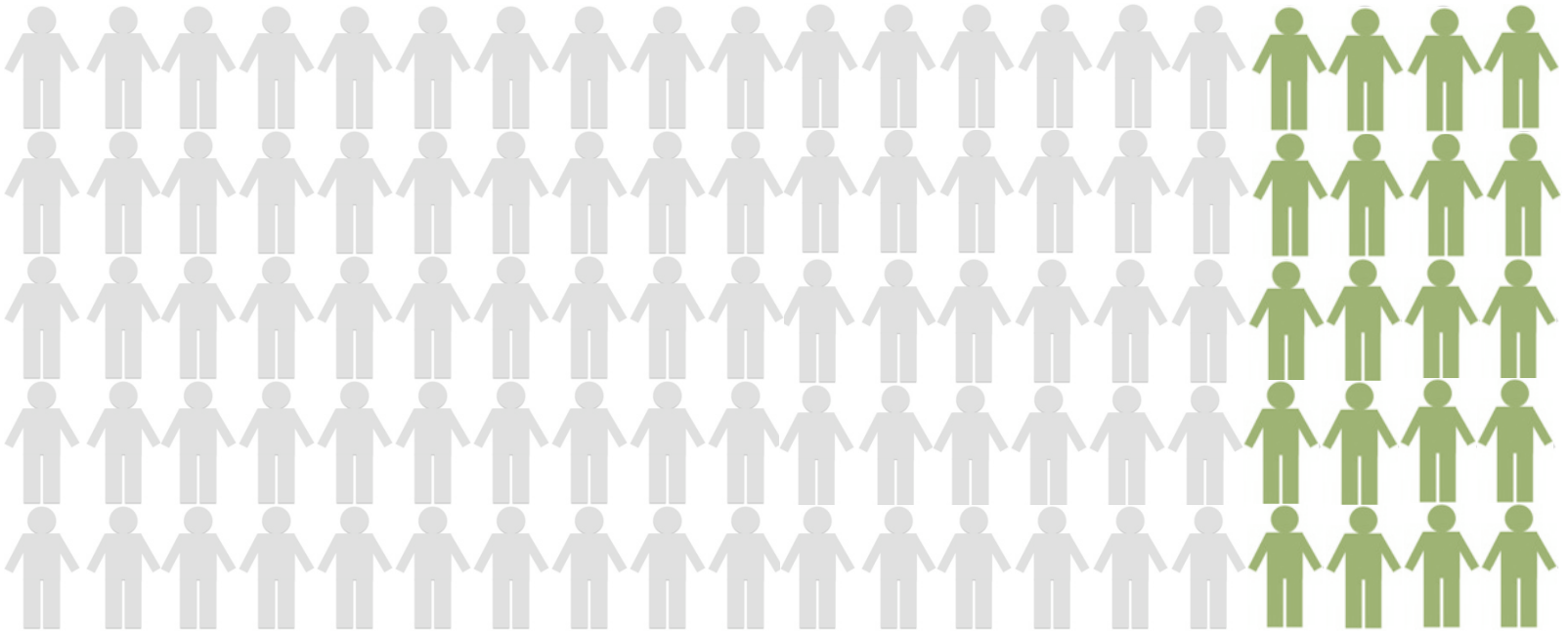
Betancourt LM et al. Adolescents with and without gestational cocaine exposure: Longitudinal analysis of inhibitory control, memory and receptive language. *Neurotoxicol Teratol* 2011; 33(1): 36-46.

Principle 6. The magnitude of an effect of exposure on disease is dependent on the prevalence of the factors that interact with that exposure

How much of our cognitive ability is determined by our genes?



= GE+



= ENV+

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Scenario 1

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Scenario 1

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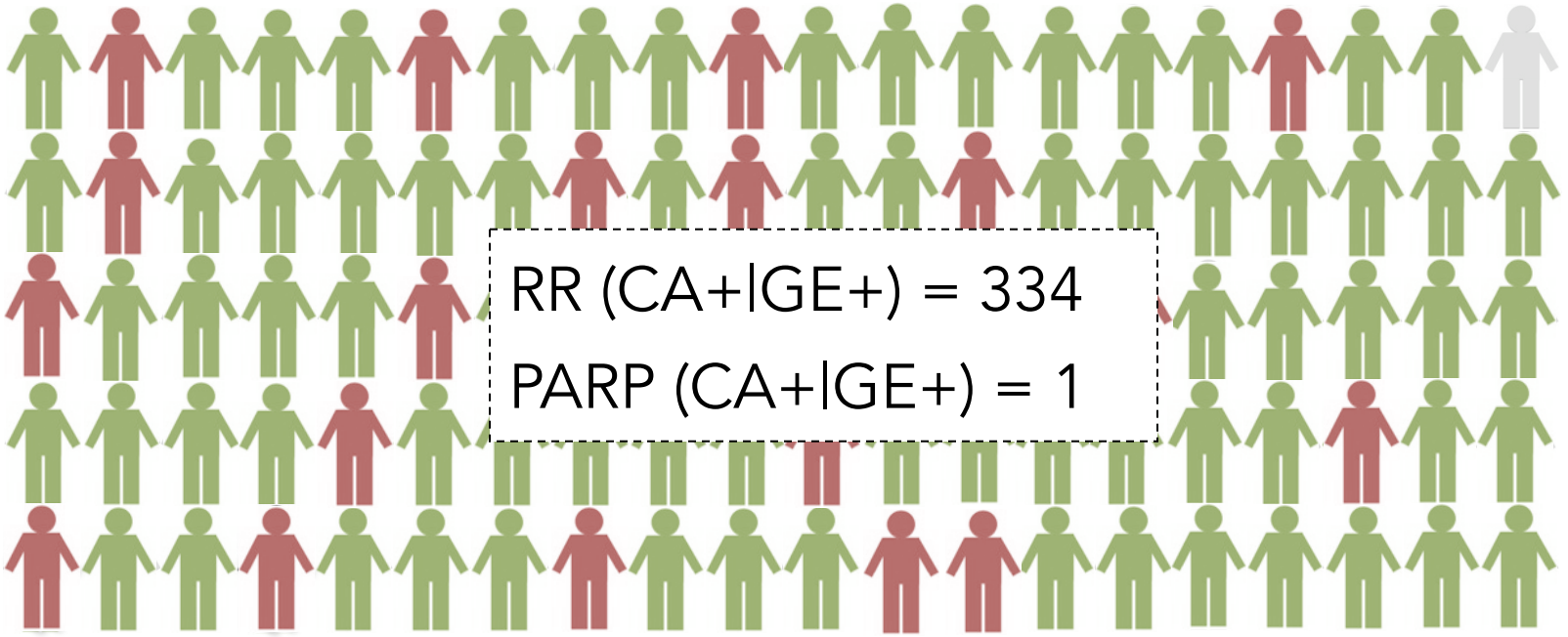
Scenario 1



= GE+ = CA+ = ENV+

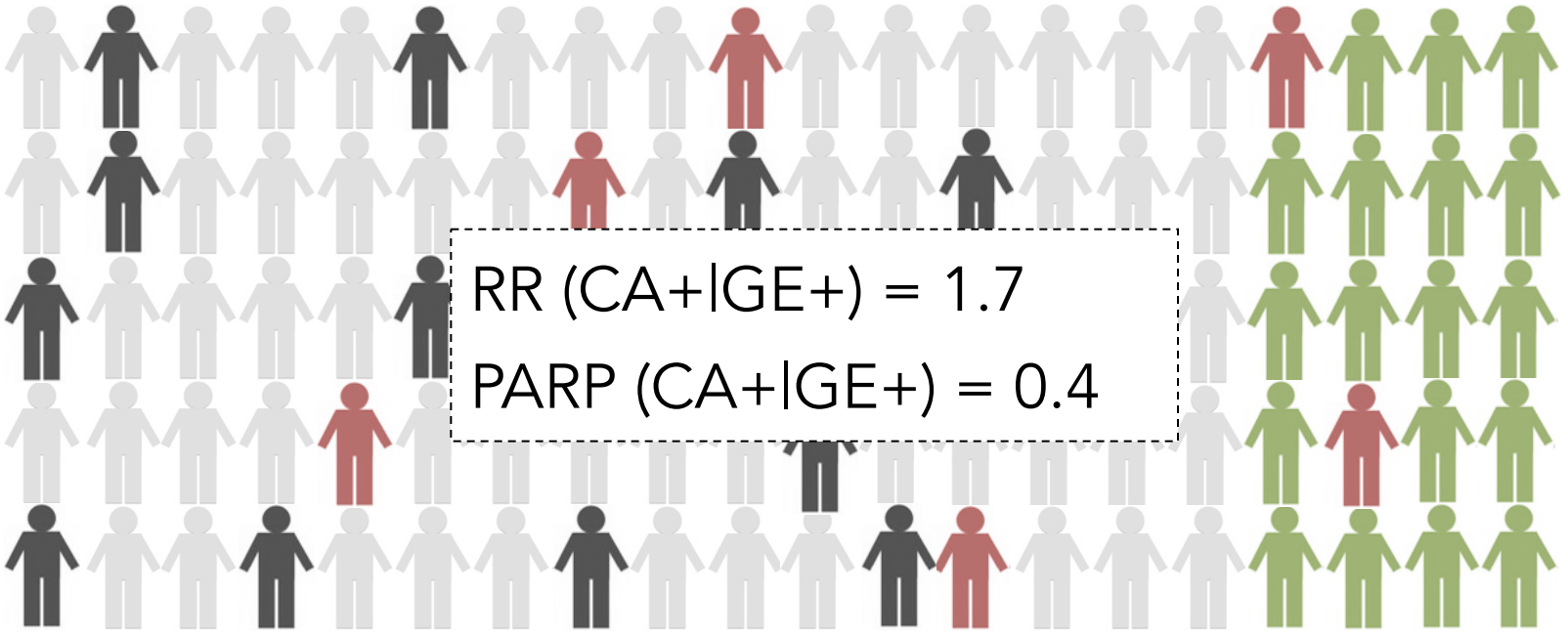
Scenario 2

= GE+ = CA+ = ENV+

Scenario 2

= GE+ = CA+ = ENV+

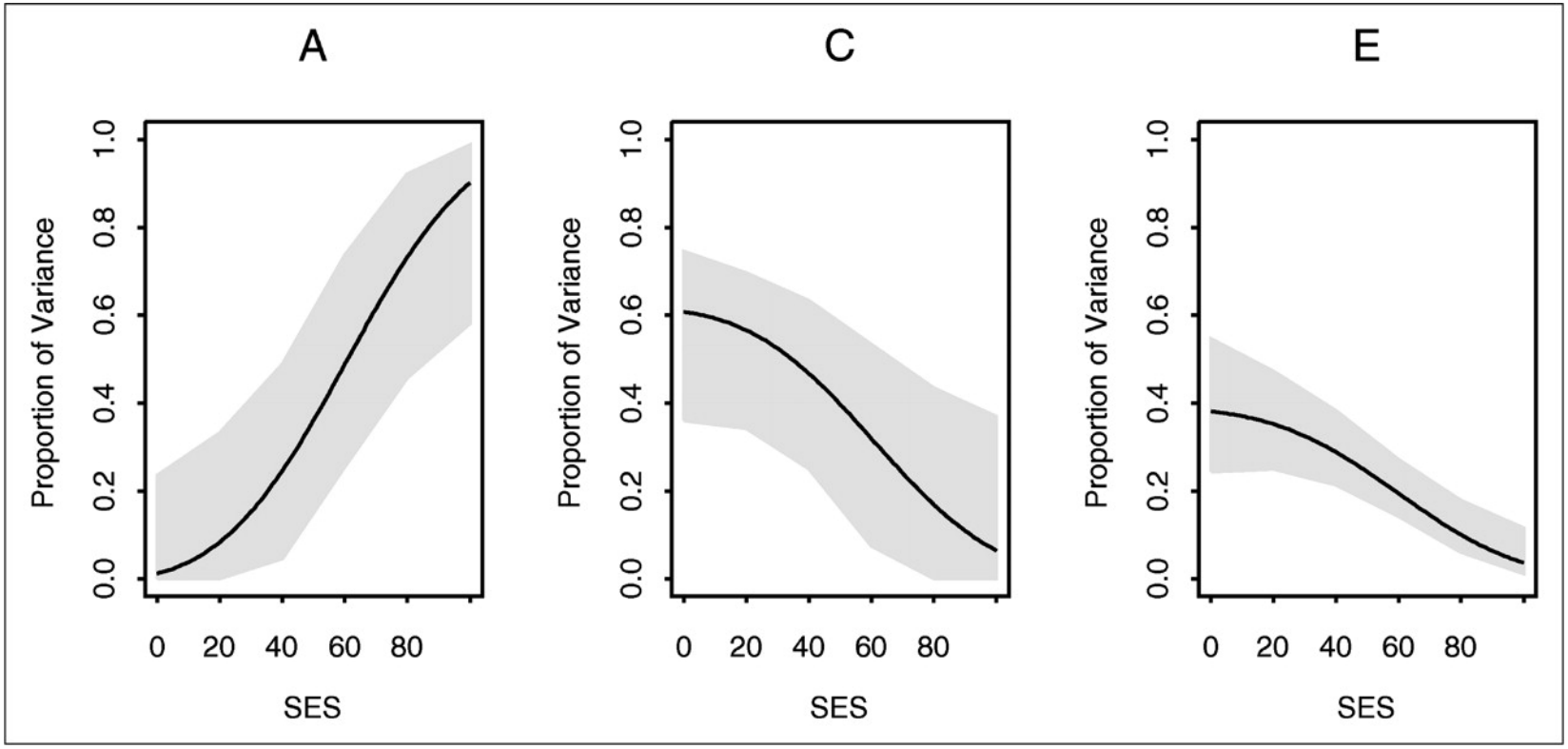
Scenario 2



= GE+ = CA+ = ENV+

Is this all theoretical?

Proportion of total Full-Scale IQ variance accounted for by A, C, and E plotted as a function of observed socioeconomic status (SES).



Turkheimer E et al. Psychological Science 2003;14:623-628

Principle 9. We can predict health in populations with much more certainty than we can predict health in individuals

Table 1. Childhood poverty as a risk factor for adult heart disease by age 65 in a population of 1,000 individuals, Population 1

	Heart disease	No heart disease	Total
Childhood poverty	25	175	200
No childhood poverty	50	750	800
	100	900	1000

$$\text{Risk ratio: } \frac{\left(\frac{25}{200}\right)}{\left(\frac{50}{800}\right)} = 2.0$$

$$\text{Positive predictive value } = \frac{25}{200} = 0.125 \text{ or } 12.5\%$$

$$\text{Negative predictive value } = \frac{750}{800} = 0.9375 \text{ or } 93.8\%$$

Table 2. Childhood poverty as a risk factor for adult heart disease by age 65 in a population of 1,000 individuals, Population 2

	Heart disease	No heart disease	Total
Childhood poverty	100	100	200
No childhood poverty	200	600	800
	300	700	1000

$$\text{Risk ratio} = \frac{\frac{100}{200}}{\frac{200}{800}} = 2.0$$

$$\text{Positive predictive value} = \frac{100}{200} = 0.50 \text{ or } 50\%$$

$$\text{Negative predictive value} = \frac{600}{800} = 0.75 \text{ or } 75\%$$

Table 2. Childhood poverty as a risk factor for adult heart disease by age 65 in a population of 1,000 individuals, Population 2

	Heart disease	No heart disease	Total
Childhood poverty	100	100	200
No childhood poverty	200	600	800
	300	700	1000

$$\text{Risk ratio} = \frac{\frac{100}{200}}{\frac{200}{800}} = 2.0$$

While strength of association stays the same, predictive value changes as prevalence of heart disease increases from 10% in Population 1 to 30% in Population 2

The Telegraph

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HOME » NEWS » WORLD NEWS » US ELECTION

Nate Silver: politics 'geek' hailed for Barack Obama wins US election forecast

An American political blogger, Nate Silver, has emerged as the other major winner during the US election for predicting all major results and Barack Obama's victory.

Image 1 of 2

Nate Silver, a former economist, writes the FiveThirtyEight blog for the New York Times. Photo: GETTY IMAGES

VIDEO
Romney concedes defeat

VIDEO
Obama's victory speech in full

VIDEO
Obama supporters party through night

By Andrew Hough
6:30PM GMT 07 Nov 2012

Follow 4,508 followers

Nate Silver, who writes the **FiveThirtyEight blog for the New York Times**, basked in worldwide adulation, after his polling correctly forecast the presidential outcome in 49 states.

The statistician, who tracked the president's statistical odds, will have correctly foreseen the result in every US state if Mr Obama's lead is maintained in Florida.

On election day, the 34 year-old, who had became a target for conservatives, also offered a 90.9 per cent probability of an Obama win in the US election.

He forecast that Mr Obama would win 332 electoral college votes compared to 206 for Mr Romney, which will be correct if the Democratic candidate wins Florida.

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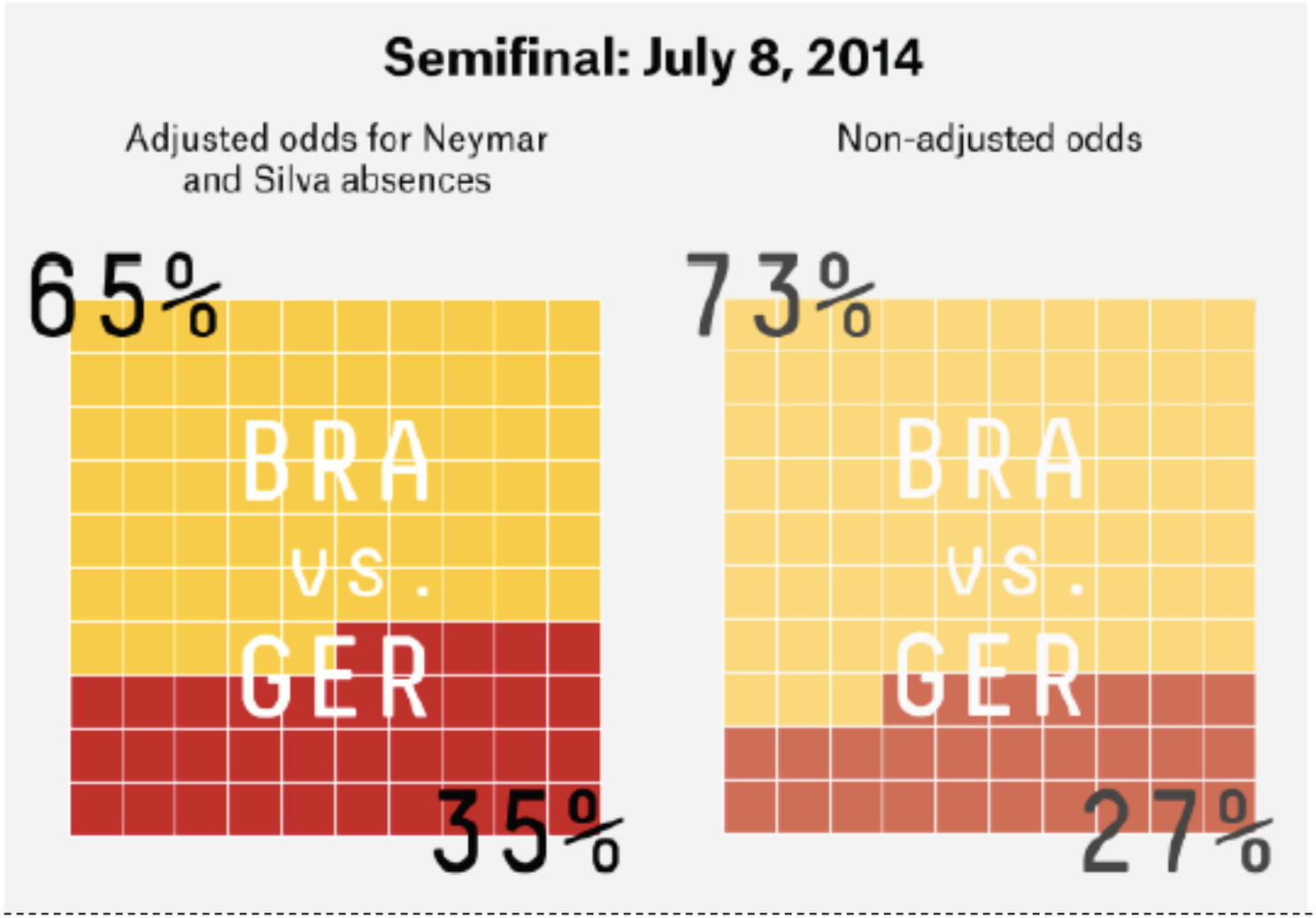

http://fivethirtyeight.com/datalab/world-cup-semifinal-crib-notes-brazil-vs-germany/ Page

Brazil crushed by Germany in the most humiliating defeat in World Cup history

1-7

Floored: the Brazil defence look shocked as Thomas Muller puts Germany ahead AP

MARTIN SAMUEL
Chief Sports Writer
reports from Belo Horizonte

THERE must have been a moment when even those watching in their white Mannschaft shirts inside Estadio Mineirao wished it would end. Germans are only human, after all.

Admittedly, the 11 on the pitch seemed to be transcending that state at times, but those looking on, the mortals — they must have felt it like the rest of us.

It was simply too much. Too brutal. Too excruciating. Too

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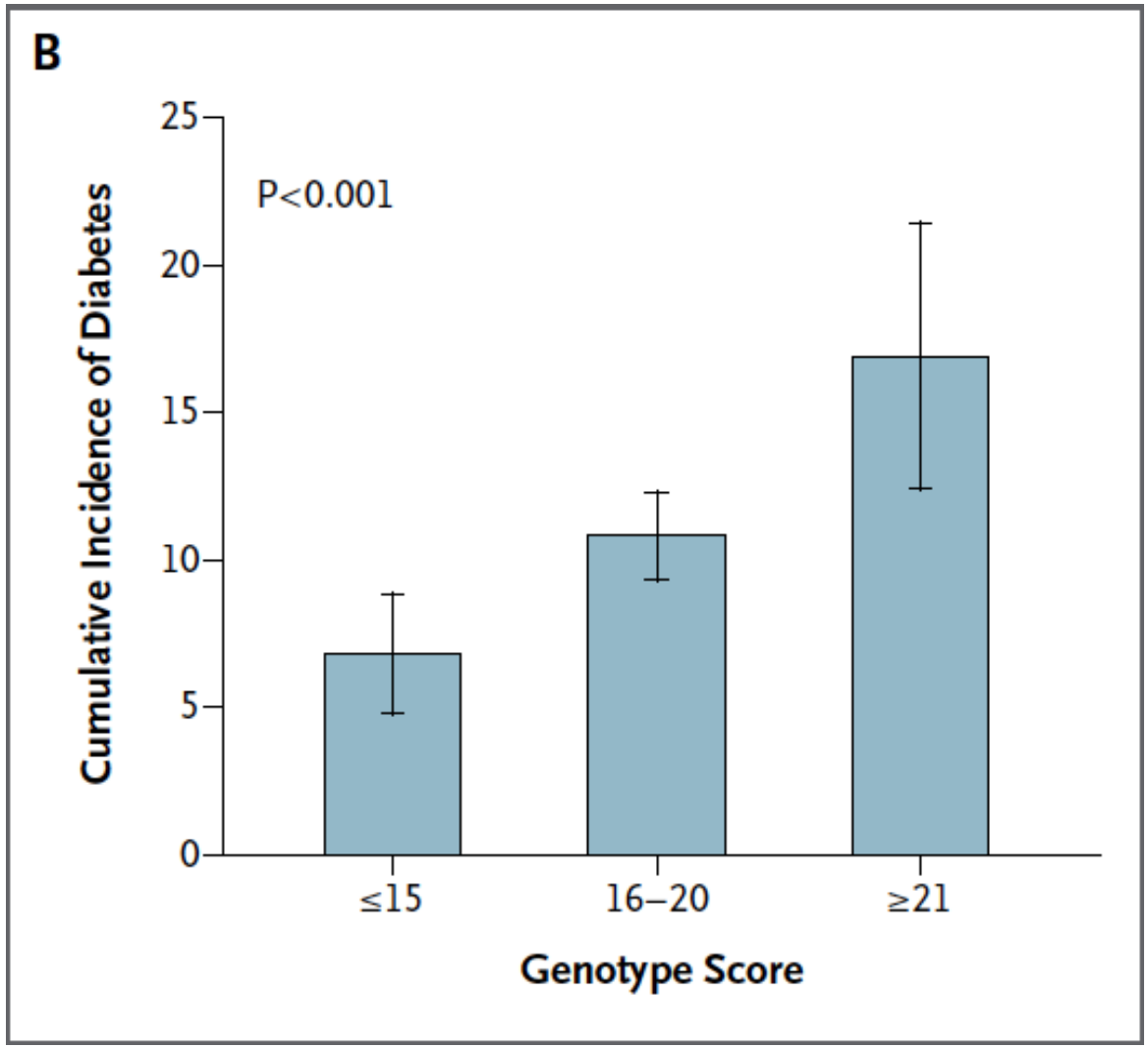
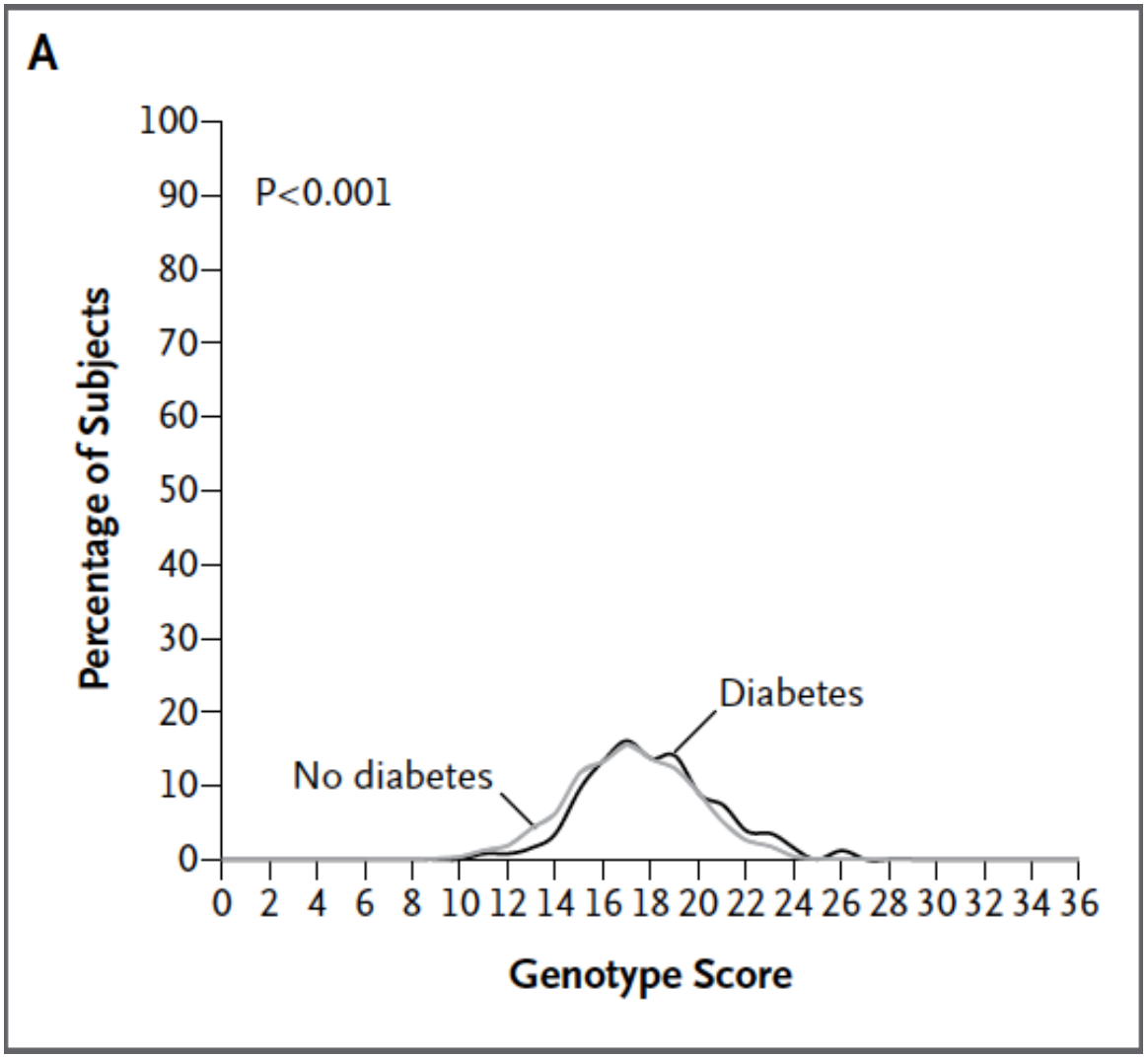
IMPORTANT INFORMATION

NEGLIGENCE AT HOSPITAL OR DOCTORS?

Compensation could be available



Meigs JB, Shrader P, Sullivan LM, McAteer JB, Fox CS, Dupuis J, Manning AK, Florez JC, Wilson PW, D'Agostino RB Sr, Cupples LA. Genotype score in addition to common risk factors for prediction of type 2 diabetes. N Engl J Med. 2008 Nov 20;359(21):2208-19



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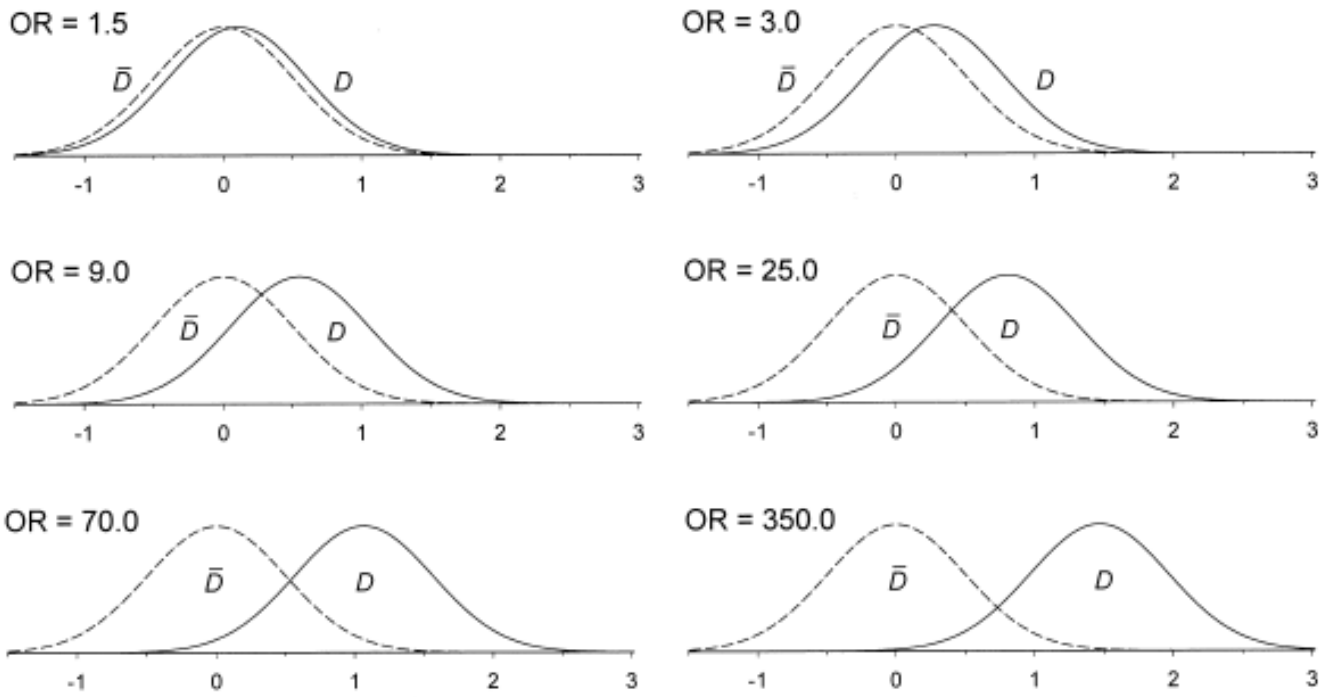

FIGURE 2. Probability distributions of a marker, X, in cases (solid curves) and controls (dashed curves) consistent with the logistic model logit$P(D = 1|X) = \alpha + \beta X$. It has been assumed that X has a mean of 0 and a standard deviation of 0.5 in controls so that a unit increase represents the difference between the 84th and 16th percentiles of X in controls. The marker is normally distributed, with the same variance in cases. The odds ratio (OR) per unit increase in X is shown.

Pepe MS, Janes H, Longton G, Leisenring W, Newcomb P. Limitations of the odds ratio in gauging the performance of a diagnostic, prognostic, or screening marker. American Journal of Epidemiology 2004; 159:882-890.

1. Population health manifests as a continuum.
2. The causes of differences in health across populations are not necessarily an aggregate of the causes of differences in health within populations.
3. Large benefits to population health may not improve the lives of all individuals.
4. The causes of population health are multilevel, accumulate throughout the life course, and are embedded in dynamic interpersonal relationships.
5. Small changes in ubiquitous causes may result in more substantial change in the health of populations than larger changes in rarer causes.
6. The magnitude of an effect of exposure on disease is dependent on the prevalence of the factors that interact with that exposure.
7. Prevention of disease often yields a greater return on investment than curing disease after it has started.
8. Efforts to improve overall population health may be a disadvantage to some groups; whether equity or efficiency is preferable is a matter of values.
9. We can predict health in populations with much more certainty than we can predict health in individuals.

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